

**Prevalence and Echocardiographic Quantification
of Mitral Regurgitation including 3D-EF [Ejection
fraction] assessment in Acute ST Elevation
Myocardial Infarction**

A dissertation submitted in partial fulfillment of
DM- Branch II Cardiology Examination of
the Tamilnadu Dr.MGR Medical University, Chennai,
to be held in July/August 2013

BONAFIDE CERTIFICATE

This is to certify that the work presented in this dissertation titled **“Prevalence and Echocardiographic Quantification of Mitral Regurgitation including 3D-EF[Ejection fraction] assessment in Acute Myocardial Infarction”** done towards fulfillment of the requirements of The Tamilnadu Dr. M.G.R. Medical University, Chennai for the DM – branch II cardiology examination to be conducted in July/August 2013, is a original work of the candidate Dr. Amit Hooda, Post-graduate student in the department of cardiology, Christian Medical College, Vellore under my guidance and supervision. This dissertation has not been submitted, fully or in part to any other board or university.

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DECLARATION

I, Dr. Amit Hooda, solemnly declare that this dissertation entitled, **“Prevalence and Echocardiographic Quantification of Mitral Regurgitation including 3D-EF [Ejection fraction] assessment in Acute ST Elevation Myocardial Infarction”** is a bonafide work done by me at the department of Cardiology, Christian Medical College, Vellore under the direct guidance and supervision of Dr. V Jacob Jose, Professor, Department of Cardiology, Christian Medical College, Vellore and my co-guide Dr. Purendra Kumar Pati, Professor, Department of Cardiology, Christian Medical College, Vellore.

This dissertation has not been submitted by me either in part or in full on any previous occasion to any university or institution for the award of any degree or diploma.

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CONTENTS

Abstract

Page No.

• Introduction	...1
• Aim and Objectives	...3
• Review of Literature	...4
• Methodology	...33
• Results	...36
• Discussion	...49
• Limitations	...53
• Conclusions	...54
• Bibliography	...55
• Annexure	
○ Information sheet	
○ Study Proforma	
○ Glossary for master data chart	
○ Master data chart	

*Prevalence and Echocardiographic
Quantification of Mitral Regurgitation
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Myocardial Infarction*

Title of the abstract: **PREVALENCE AND ECHOCARDIOGRAPHIC ASSESSMENT OF MITRAL REGURGITATION INCLUDING 3D-EF [EJECTION FRACTION] ASSESSMENT IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION**

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OBJECTIVES

This study intended to assess the prevalence of Mitral Regurgitation in patients of acute ST elevation Myocardial Infarction (STEMI) during index hospitalization and during 1 month follow up along with 3D EF assessment.

METHODS

119 consecutive patients with STEMI were assessed for mitral regurgitation using various quantitative echocardiographic parameters like jet width, vena contracta and PISA during index hospitalization as well as during 1 month follow up. Left ventricular ejection fraction was assessed by both Simpson's method as well as 3D echocardiography. All continuous variables are expressed as mean \pm SD and categorical variables as number (percentages). Independent samples T test was used for comparative analysis of two continuous variables. Comparison of categorical variables was done using Chi square test. Pearson correlation coefficient was used to analyze the correlation between two continuous variables.

RESULTS

STEMI patients have 22.7% prevalence of MR at onset, which is more likely in older age group, diabetics and IWMi patients and independent of gender and left ventricle systolic dysfunction. Most commonly, ischaemic MR is of milder severity at onset which tends to persist in 15.7% of patients during follow up. Some patients may develop new MR later on depending on the LV remodeling. Modalities of revascularization doesn't influence the course of Mitral Regurgitation in short term follow up. 2D EF estimation with a properly acquired good image is comparable to more precise 3D EF in STEMI patients.

INTRODUCTION

MR is a frequent complication of coronary artery disease. It was often under-rated because it is clinically silent, but with the use of echocardiography this complication is observed between 15%-20% after a myocardial infarction.¹ When compared with patients without MR, the patients with acute myocardial infarction and mitral regurgitation are older, more often in female and frequently have a history of previous ischaemic heart disease.² Its presence and degree have major prognostic implications and underscore the importance of its detection and quantification. Ischaemic mitral regurgitation can independently predict cardiovascular death with a relative risk of 2.³ In a community trial, Bursi et al found that mitral regurgitation predicted heart failure as well as mortality among 1 month survivors independent of age, gender, ejection fraction, and Killip Class.⁴ Even mild MR was shown to increase the mortality in SAVE trial (Survival and Ventricular Enlargement)³ hence, detection and quantification of MR is crucial.

MR can be estimated by different techniques on echocardiography. Color flow imaging allows an easy visualization of MR, but was found to overestimate the MR.⁵ McCully et al demonstrated that the same jet area corresponds to smaller regurgitant volumes in functional MR as compared to organic MR.⁵ Doppler echocardiography allows accurate assessment of regurgitant volume and effective regurgitant orifice and thus provide the tools to reliably

evaluate the prognosis and mechanism. Doppler methods are simple, fast, reproducible and proven to be more reliable but sparsely applied in routine clinical evaluation. Current guidelines advocate quantification of MR by the measurement of vena contracta and proximal isovelocity surface area (PISA), the most recommended quantitative approach whenever feasible. The semi-quantitative evaluation should be abandoned.⁶

Echocardiography is the most common method to assess left ventricular systolic function. It can efficiently predict the outcome and help in determining the treatment modalities like CRT-D implantation etc. Routine 2D EF measurement has several limitations in AMI patients due to problems of foreshortening and geometric assumptions. 3D echo LVEF is much more accurate especially in presence of regional wall-motion abnormalities as it does not have geometric assumptions and is found to have comparable with present day “gold standard” for systolic function, cardiac MRI.⁷ It can automatically calculate ejection fraction and left ventricular mass using the automated software's, and is therefore more reproducible. It is several folds accurate as compared to conventional echo.

As there is no data for prevalence of Mitral Regurgitation in Indian population, this study was undertaken. We evaluated the prevalence and degree of Mitral Regurgitation in the acute phase and after 1 month of STEMI.

AIMS AND OBJECTIVES

PRIMARY AIM

- a. To study the following parameters in Acute ST Elevation Myocardial Infarction[STEMI] patients
 - i. Prevalence of mitral regurgitation[MR] by echocardiography in acute STEMI and during 1 month follow up
 - ii. 3D-EF(ejection fraction) assessment

SECONDARY AIM

- a. Correlation of 2D-EF, 3D-EF and MR in STEMI patients

REVIEW OF THE LITERATURE

INTRODUCTION

Mitral regurgitation (MR) is defined as systolic retrograde flow from left ventricle (LV) to left atrium (LA) because of pressure gradient between the two chambers.⁸

The term ischaemic MR does not necessarily imply the presence of true myocardial ischemia, it is in fact an abridgment, characterizing a clinical situation corresponding to chronic coronary artery disease with frequently a prior history of one or more myocardial infarctions leading to progressive global or regional pathological LV remodelling, usually in the absence of reversible ischaemia.⁹

Ischemic MR is a type of secondary/functional MR due to coronary artery disease. Secondary MR is defined as functional MR, due to LV remodelling by idiopathic cardiomyopathy or coronary artery disease.^{8, 10} It is important to distinguish between primary MR due to organic disease of one or more components of the mitral valve apparatus and secondary MR which is not a valve disease, but represents the valvular consequences of a LV disease. There are however limitations in both terms: functional and ischemic. Indeed, recent studies have demonstrated evidence of structural changes in the mitral leaflets in response

to tethering on them by LV pathological remodelling. The leaflet adaptation includes enlargement and increased stiffness.¹¹

PREVALENCE

MR is a frequent accompaniment of ischaemic heart disease. Clinical presentation is variable from silent to severe MR presenting with hemodynamic instability. It may be an incidental finding on echocardiography or catheterization.³ Its importance was often underscored because of low murmur intensity but with the use of echocardiography, MR is observed between 15%-20% of patients with acute myocardial infarction.¹ Different investigators have found variable incidence of MR in acute MI with the help of different imaging modalities. Lehmann et al found 13 % incidence of mitral regurgitation early in the course of acute myocardial infarction with the help of contrast left ventriculography.² Tchong et al reported incidence of post-infarction mitral regurgitation was to be 17.9% of patients within hours of infarction.¹² Indeed, when it is sought by doppler, MR has been reported to occur in up to 39% of patients with MI.^{13,14} With the recent advances in non-invasive doppler echocardiography, it is possible to accurately assess the regurgitant volume and effective regurgitant orifice. Old age, diabetes, past history of MI, severe CAD are more frequently associated with STEMI with

MR than patients with STEMI without MR.^{2, 12, 15} Its presence and degree have major prognostic implications and mandates its detection and quantification.

PATHOPHYSIOLOGY

Normal mitral valve function depends on perfect delicate interplay between the mitral leaflets, the subvalvular- apparatus (which includes: chordae tendinae and papillary muscle), the mitral annulus, and the left ventricle. Pathophysiology of ischemic MR is perplexing. Myocardial damage and LV dysfunction usually precedes MR. Ischemic MR is characterized by normal leaflets and subvalvular apparatus and occurs due to restricted motion of the leaflets. According to the Carpentier's classification, ischaemic mitral regurgitation is classified as type 3.¹⁶

Given below is the classification based on motion of leaflet in relation to the mitral annular plane (figure 1)

1: normal leaflet motion. Perforation of the leaflet due to traumatic injury or endocarditis, or annular dilatation, which may cause left ventricular disease, is the cause of MR in type1.

2: excessive leaflet mobility accompanied by displacement of the free edge of one or both leaflets beyond the mitral annular plane into the left atrium, degenerative cardiac diseases may cause leaflet prolapse.

3: leaflet restriction. It is further subclassified into 2 varieties:

3a, where the restriction occurs throughout the cardiac cycle, i.e. both in systole and diastole due to shortening of the chordae and/or leaflet thickening such as in rheumatic disease,

3b, where the leaflet restriction is seen in systole only (usually the result of regional wall motion abnormalities seen in ischaemic mitral regurgitation).¹⁶

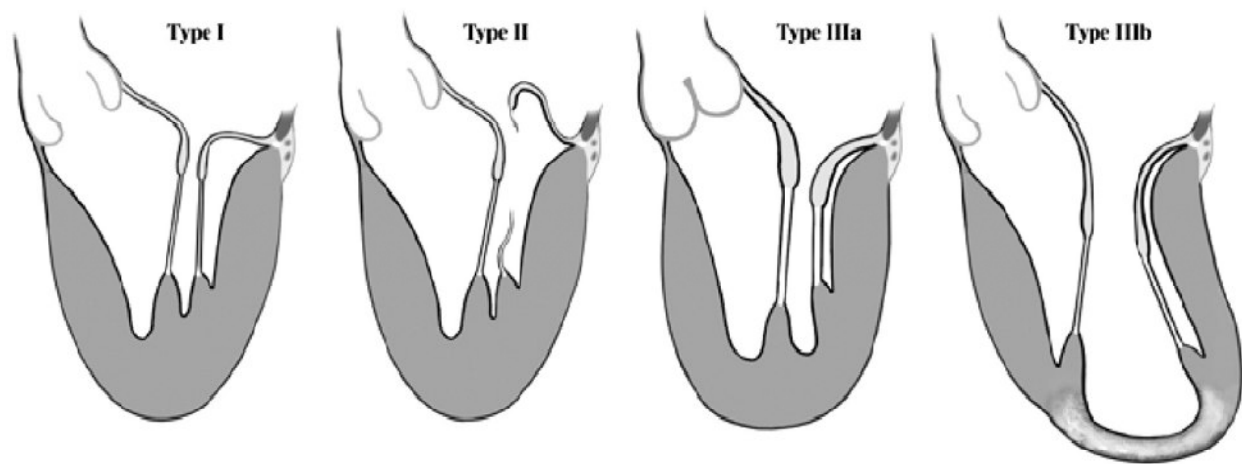


Figure 1: Carpentier's functional classification of Mitral Regurgitation¹⁷

Mitral Leaflet closure is mainly an intricate interplay between the forces of tethering and ventricular forces (figure 2, 3). Increase in tethering forces will not allow adequate closure of the mitral leaflets.^{8, 18} The most frequent pattern seen is posterior infarction, more commonly transmural, leading to local left ventricular pathological remodelling and thereby contributing to posterior, apical and lateral displacement of the posterior papillary muscle. As the papillary muscle extends non-extensible chordae to both the leaflets, its displacement results in a more apical tethering of the leaflets and their coaptation point, and a characteristic deformity of the anterior leaflet which is described as the 'seagull sign'.^{8, 19} The tethering process produces the shape like that of a tent between the annular plane and the displaced leaflets. The tenting volume closely estimates the regurgitant orifice area.^{8,20} Tenting area is asymmetric in case of posterior infarction and regional remodelling, predominates on the posterior leaflet close to the medial commissure. It is accompanied by decreased mobility of the posterior leaflet. In another subgroup of patients with previous anterior infarction or both anterior and posterior infarctions, LV dilatation is more global, LV is more spherical, both papillary muscles are displaced, the tenting area is symmetric, the regurgitant jet is central and the contribution of annular dilatation and flattening is more important. Second important determinant is decrease in ventricular closing forces, includes altered systolic annular contraction, LV dysfunction, reduced synchronicity

between the two papillary muscles and global LV dyssynchrony, especially in basal segments.⁸ Factors aggravating the mitral regurgitation are dilatation of the mitral annulus and the decrease in systolic annular contraction, but isolated annular dilatation does not create functional mitral regurgitation.¹⁸

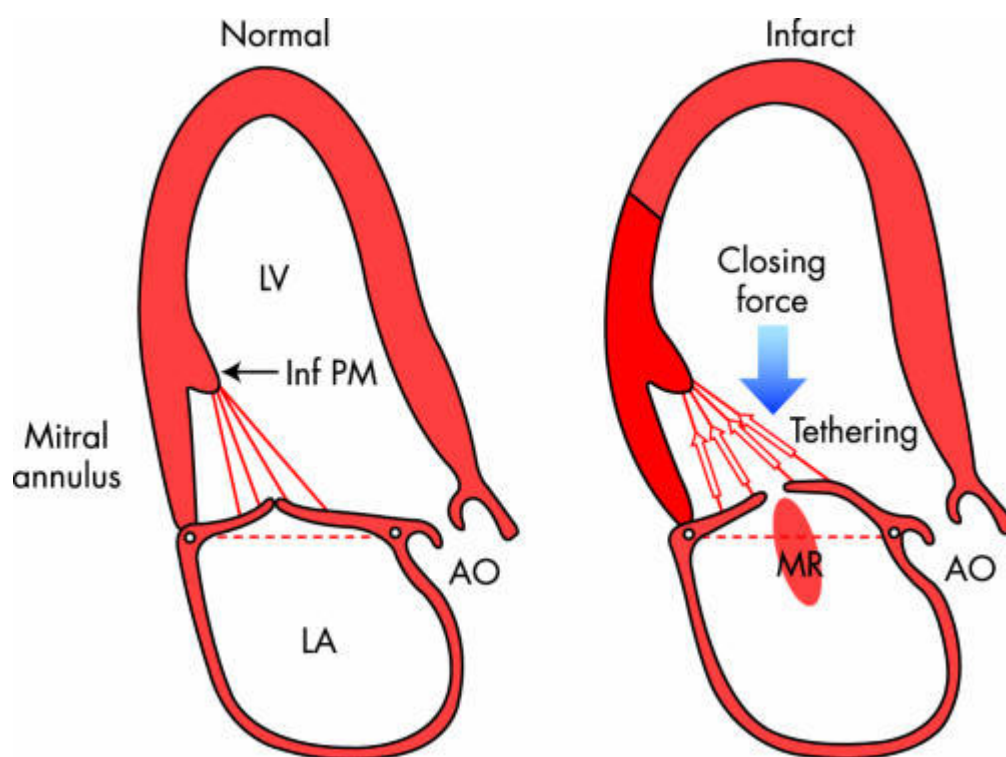


Figure 2: Left figure: Normal coaptation is seen. **Right figure:** tethering of leaflet due to papillary muscle displacement and annular dilatation

AO, aorta; Inf PM, inferior papillary muscle; LA, left atrium; LV, left ventricle; MR, mitral regurgitation.

(Reproduced from Levine RA, Hung J, Otsuji Y, et al. *Mechanistic insights into functional mitral regurgitation*. Curr Cardiol Rep 2002; 4:125–9)

Occasionally, leaflet prolapse can occur as a result of fibrotic elongation of papillary muscle which may be followed by an event such as myocardial infarction and this can result in MR. (Carpenters' Type 2).

The consequences of MR depend on the following underlying factors

- a. Severity of regurgitation
- b. LA compliance
- c. LV-LA gradient (the driving force)
- d. Duration of the lesion

Acute MR can occur secondary to two rare causes: rupture of a papillary muscles resulting from acute myocardial infarction and transient active ischemia leading to true ischemic MR. The rupture of a papillary muscle, more often in the location of head of the postero-medial papillary muscle, is a catastrophic complication of actual MI with a high mortality if emergency surgery is not done.^{8, 21}

In majority of patients with chronic ischemic MR complicating left ventricular dysfunction and heart failure, LA is enlarged and has a greater compliance with

low driving force. The volume overload resulting from MR contributes to a vicious cycle : the more remodelling of left ventricle, the more severe MR begets MR, so greater the severity of MR, larger the volume overload on LV which finally leads to LV remodelling. LV becomes more and more dilated and spherical in accordance to laplace law, which further propentiates the MR. Despite a reduction in LV impedance, LV wall stress increases ,finally translating into LV dysfunction.²² Chronic ischaemic MR finally leads to development of pulmonary arterial hypertension.

Another important feature of ischaemic MR is its dynamicity.²³ The degree of MR is best quantified by the effective regurgitant orifice (ERO) area.²⁴ The regurgitation area keeps on changing throughout the systole due to dynamic changes in transmitral pressure, though it is of lesser importance in mid-systole when compared to other phases of systole.^{25,26} ERO is load dependent, therefore affected by daily activities. Another evidence of the dynamicity of ischaemic MR is a reduction of regurgitant volume related to a reverse LV remodelling obtained by appropriate medical treatment.²⁷ Dynamic nature of MR can be very well appreciated during an exercise doppler echocardiogram.²⁸ The degree of MR seen at rest is not related to exercise-induced changes in ERO area or regurgitant volume.²⁹ Exercise-induced changes are quite variable in different individuals.

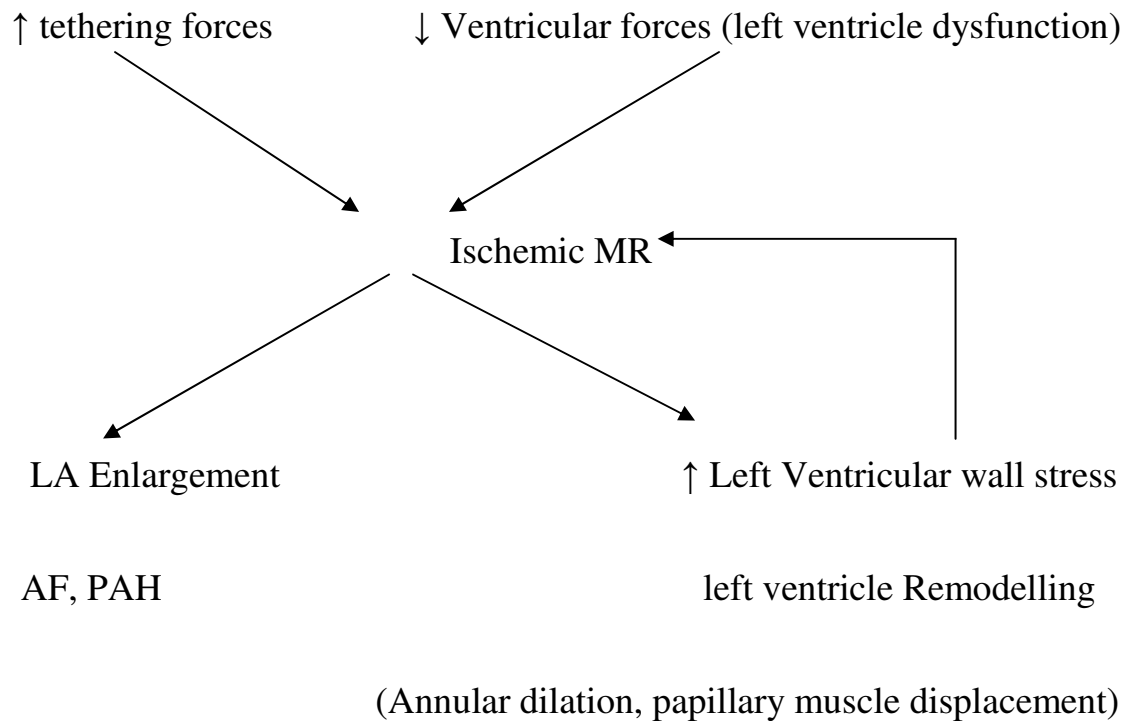


Figure 3: Pathophysiology of Ischaemic MR

In some individuals with moderate or severe MR at rest, a decrease in ERO area can be observed with exercise and usually results from contractile reserve of the LV, in particular of the postero-basal segment and/or a reduction in intra LV dyssynchrony.³⁰ Around 30% of these patients develop an increase in the severity of regurgitation and in systolic pulmonary artery pressure during exercise. The degree of exercise-induced increase or decrease in MR relates to changes in valve deformation, LV remodelling and papillary muscles synchronicity.

DIAGNOSIS AND QUANTIFICATION

Clinical examination in ischaemic MR may reveal a regurgitant murmur of low grade or may even be silent or inaudible .It is an insensitive method for ischaemic MR diagnosis due to subtle or near normal auscultatory findings in many patients. This can be explained from the fact that cardiac output is low, LV contractility is compromised and atrial pressure is high thereby they cause lower regurgitant volume, consequently low grade murmur in ischemic MR compared to organic MR. There is no correlation between intensity of murmur and severity of ischaemic MR.

The diagnosis of ischaemic MR is usually made by using imaging modalities like doppler echocardiography. Doppler echocardiography is a useful tool in diagnosis and is superior to other techniques like contrast ventriculography. Importance of doppler echocardiography in routine clinical practice is undisputed. Clinically subtle findings like low intensity murmurs should always lead to a careful echocardiographic examination.

Quantification of MR is also crucial. Echocardiography plays a key role not only in diagnosis of regurgitant lesion but also in the assessment of the mechanism and the severity of MR. It also has a role in determining treatment options as it helps in determining the feasibility of valve repair versus replacement.

Assessment of Mitral Regurgitation by echo can be (a) qualitative (b) quantitative.

QUALITATIVE METHODS

Colour flow imaging

Colour flow imaging is the most widely used method to assess MR severity echocardiographically.³² This measurement is poorly reproducible and influenced by various factors. Though it allows an easy visualization of the regurgitation and frequency of MR, it has major limitations in assessing the severity of MR.^{5, 33} It tends to overestimate the severity of regurgitation. It is generally assumed that with increasing MR, the size and extent of the regurgitant jet in LA is increased. Larger colour jets extending deep into LA represent greater severity of MR than small thin jets that appear just beyond the mitral leaflets. However, the jet size may be influenced by other factors such as technical and hemodynamic influences and therefore caution must be exercised in interpretation of jet severity based on jet size. For the same severity of MR, patients with increased LA pressure, enlarged LA or wall hugging eccentric jet may show smaller jets area when compared with normal LA pressure, size or with central jets.⁶ In acute MR, even centrally directed jets may be misleadingly small. Nevertheless, a large eccentric jet adhering, swirling and reaching the posterior wall of the LA favours significant MR and

smaller thin jets appearing just beyond the mitral leaflets usually is an indicator of mild MR.

Continuous Wave Doppler of MR jet

Continuous wave doppler of mitral regurgitation jet is another qualitative parameter which is used to measure the severity of mitral regurgitation. The signal intensity (jet density) of the CW envelope of the mitral regurgitant jet can be used as a qualitative indicator to mitral regurgitation severity. A dense triangular mitral regurgitation signal with a full envelope indicates severe mitral regurgitation than a faint signal. Truncated (notch) envelope with a triangular contour and an early peak velocity (blunt) indicates elevated LA pressure or a prominent regurgitant pressure wave in the LA due to severe MR. In case of eccentric mitral regurgitation; it may be difficult to record the full CW envelope of the jet, while the density may be used as indicator for assessing the severity.

SEMIQUANTITATIVE MEASURES

The semi-quantitative evaluation of regurgitant jet area should not be used.⁶ It is recommended that these measures should only be used for diagnosing mitral regurgitation, and not to quantify the severity of MR. A more quantitative approach is recommended when more than a small central MR jet is seen.

Vena contracta width (VCW)

The vena contracta is the narrowest portion of the MR jet downstream from the orifice; it reflects the effective orifice area.³⁴⁻³⁶ Whenever feasible measure the dimensions of vena contracta, which can help in quantification of MR. Using a careful probe angulation and adapted Nyquist limit (colour Doppler scale) (40–70 cm/s), the vena contracta is typically imaged in a view perpendicular to the commissural line (e.g. the parasternal long-axis or the apical four chamber view) to identify the neck or the narrowest portion of the jet. This narrowest doppler colour sector scan can be coupled with the zoom mode to improve resolution and for more accurate measurement (Figure 4). It is recommended that if possible averaged measurements over at least three beats should be taken and measurements should be taken from two orthogonal planes. A vena contracta, < 3 mm is considered as an indicator of mild MR whereas a width ≥ 7 mm defines severe Mitral Regurgitation, values in between 3 to 7 mm are ambiguous and need further confirmation by a more quantitative method.⁶

The concept of vena contracta is based on the assumption that the regurgitation orifice is almost circular, this assumption holds true in cases of organic MR but in functional MR the results may not be accurate as the orifice may be non circular and elongated along the mitral coaptation line.^{37,38} Thus, it could look narrow in four-chamber view and broad in two-chamber view. Conventional 2D colour

Doppler imaging does not provide appropriate orientation of 2D scan planes to obtain an accurate cross-sectional view of the vena contracta. The vena contracta can be classically well identified in both central and eccentric jets. In case of multiple MR jets, the respective widths of the vena contracta are not additive. Such characteristics may be better appreciated and measured on 3D echocardiography. In cases of functional MR, a mean vena contracta width (four- and two chamber views) has been shown to be better correlated with calculation done using the 3D vena contracta.⁴¹ 3D echo assessment of the vena contracta is not used routinely and currently it is used for research purposes.

A mean value >8 mm on 2D echo has been reported to define severe MR for all aetiologies of MR including functional MR, though it needs confirmation in further studies.³⁹

Pulmonary venous flow Doppler

It is an additional parameter for evaluation of MR severity. Normally, if there is no diastolic dysfunction in venous flow Doppler we get a positive systolic wave (S) which is followed by a smaller diastolic wave (D). As the severity of MR increases, there is a blunting of the S wave velocity. Systolic blunting or systolic flow reversal will be seen in moderate and severe MR respectively.

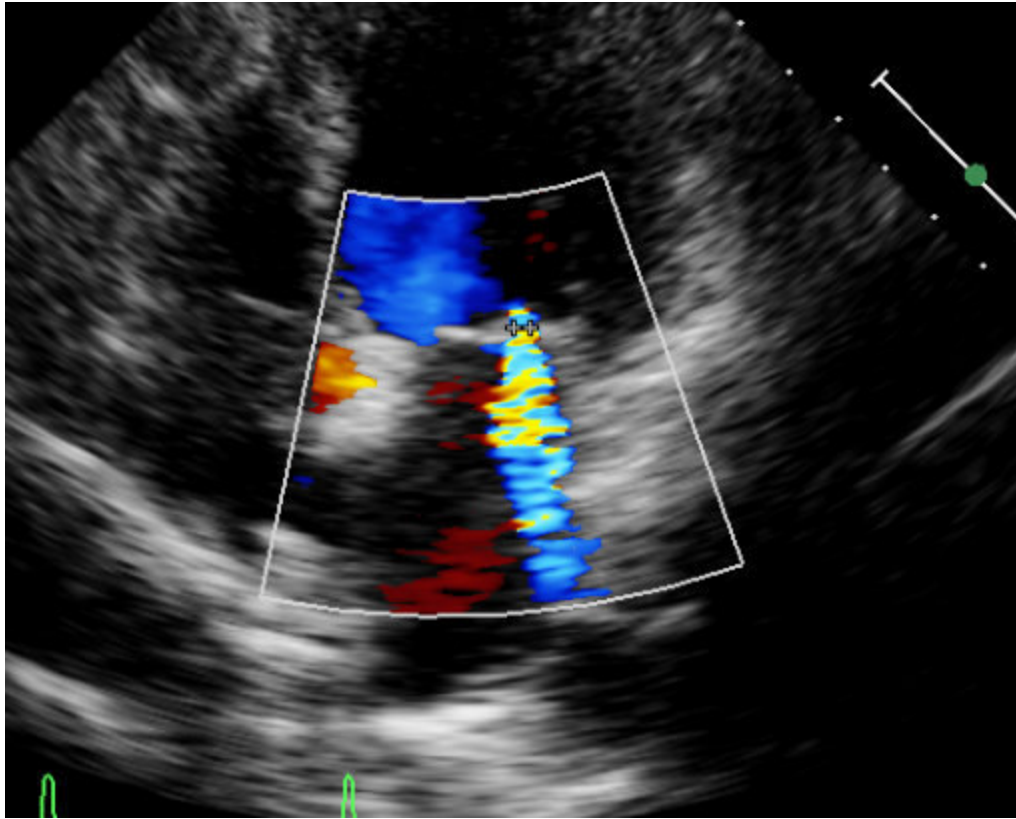


Figure 4. Measurement of Vena contracta width in Mitral Regurtiation.

QUANTITATIVE METHODS

Doppler volumetric method

Doppler volumetric method can calculate regurgitant volume by finding the difference between measured mitral and aortic stroke volume.⁴³ It can be inferred using 2D echocardiograph by calculation of LVEDV and LVESV which are calculated using biplane method of disks.⁴² Same jet areas correspond to smaller RVol in ischemic MR than in organic mitral regurgitation. However, this method is

cumbersome and time consuming. It is not recommended as first line investigation for quantification of regurgitant volume.

Flow Convergence Method

Flow-convergence method is the most frequently quantitative method for estimation of MR in current practice.⁴⁴ It is based on law of conservation of flow; its basis lies in the modified form of continuity equation. It enables the measurement of ERO area and regurgitant volume with precision.⁴⁵ As the flow convergence is proximal to the regurgitant orifice it forms the basis of analysis.⁴⁶ The apical four-chamber view is classically recommended for enabling good visualization of the proximal isovelocity surface area (figure 5). However, in anterior mitral valve prolapse the calculation of PISA should be done in the parasternal long- or short-axis view. By lowering the image depth and reducing the Nyquist limit to 15–40 cm/s, flow velocity at a hemispheric surface proximal to regurgitant orifice can be determined.⁶ The radius of the PISA is measured at mid-systole using the first aliasing. Regurgitant volume (R Vol) and effective regurgitant orifice area (EROA) are obtained using the standard formula.

Regurgitant flow = $2 \Pi (\text{radius of the flow convergence})^2 \times \text{aliasing velocity}$

ERO is the ratio of regurgitant flow /peak mitral regurgitant velocity

Regurgitant volume (RVol) is calculated as product of ERO and MR TVI.

Qualitatively, the presence of flow convergence at a Nyquist limit of 50–60 cm/s is an indicator to the presence of significant MR.⁶

Grading of organic MR ⁶

	Mild	Moderate	Severe
EROA (mm ²)	<20	20–29; 30–39	≥40
R Vol (mL)	<30	30–44; 45–59	≥60

Moderate regurgitation group can be further classified into ‘mild-to-moderate’ (EROA of 20–29 mm², R Vol of 30–44 mL) and ‘moderate-to-severe’ (EROA of 30–39 mm² or R Vol of 45–59 mL). In ischaemic MR, the thresholds of severity, which are of prognostic value, are 20 mm² and 30 mL, respectively.⁴⁷ EROA is the most robust parameter as it represents a marker of lesion severity. A large EROA can lead to large regurgitant kinetic energy (large R Vol) as well as to potential energy, with low R Vol but high LA pressure.

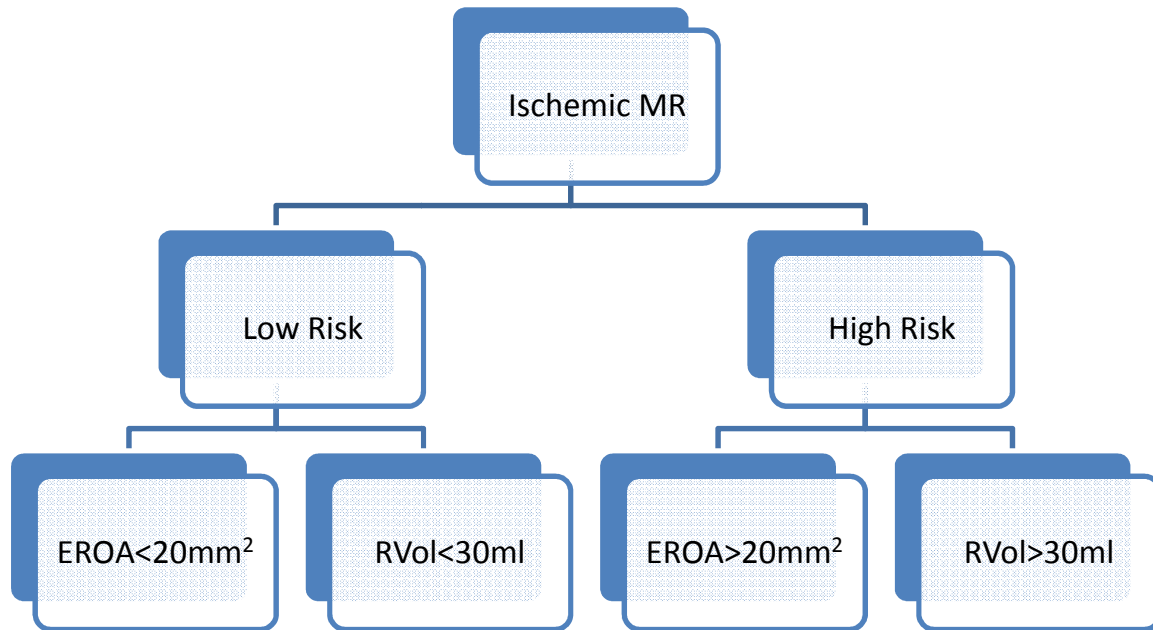


Figure 5: Ischaemic MR Grading ^(6, 48)

This method is simple, fast, and reproducible and has been validated by multiple investigators.^{46; 49} It precisely calculates the RVol, which indicates the volume overload induced by MR, and the effective regurgitant orifice (ERO), which delineates the severity of anatomic lesion.

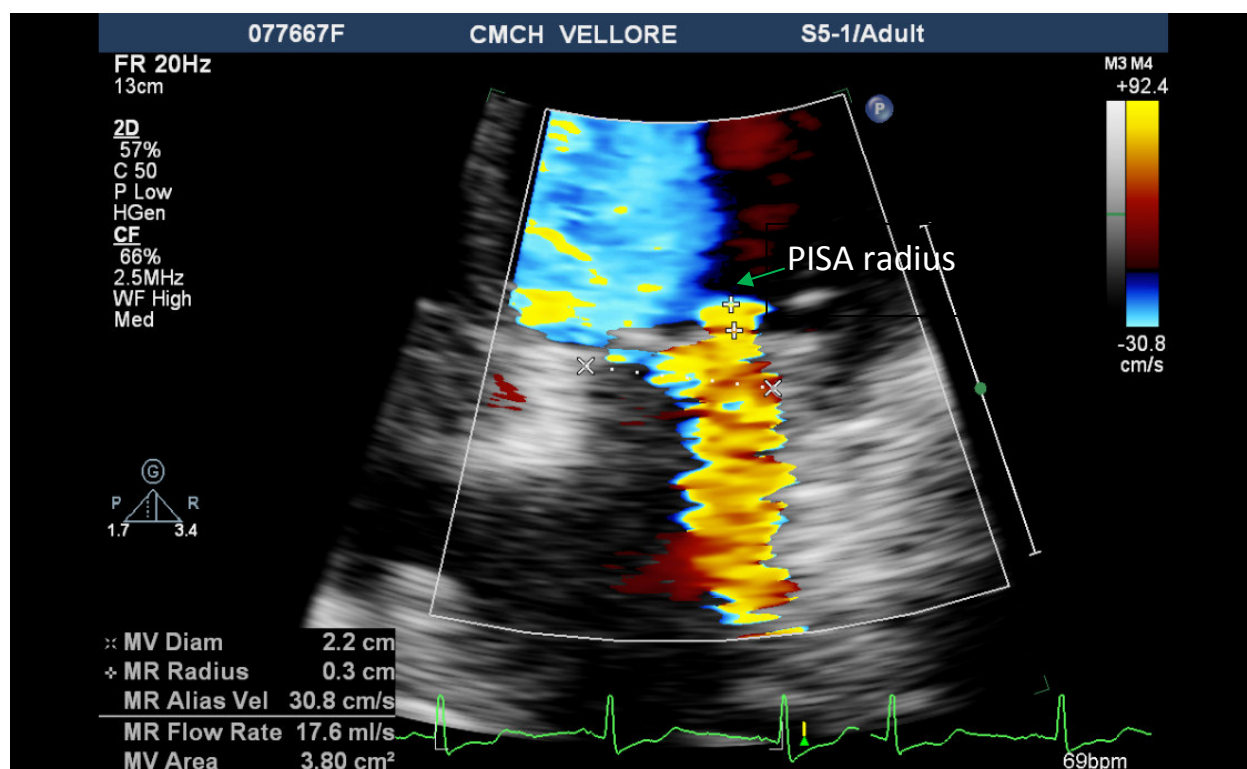


Figure 5. Determination of radius of proximal isovelocity surface area (PISA) in ischaemic Mitral Regurgitation.

There are several limitations of the proximal isovelocity surface area (PISA) approach.^{8,46} First, the PISA radius changes during systole is larger in early and late systole, and smaller in midsystole when the LV pressure is maximal.⁵⁰ Ideally, the PISA radius should not be measured at only one time point, but averaged through systole. Second, for an accurate measurement, the flow convergence should be hemispheric. In cases of functional MR, the flow convergence—a three-dimensional structure—is frequently hemielliptic (Fig 6), implying an underestimated calculation of ERO and regurgitant volume, particularly when the

ratio of long-axis length to short-axis length of the 3D regurgitant orifice is >1.5 .^{38,}

51-52

Third, multiple jets can be present; the addition of several flow-convergence regions has not been validated. Fourth, it is more accurate for central jet. It may not hold for eccentric jets, several jets, or complex or elliptical regurgitant orifices. Practically, the geometry of the PISA varies based on the shape of the orifice and mitral valve leaflets surrounding the orifice.

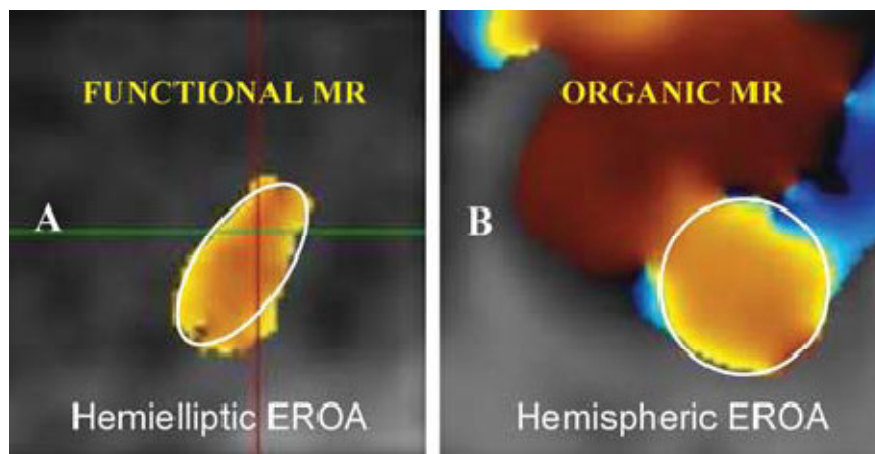


Figure 6: 3Dimensional shape of the convergence flow in (a) functional MR (b) organic MR

(Reproduced from EHJ guidelines for the assessment of the valvular regurgitation 2010)

Thus, practically, the most reliable calculation of regurgitant volume and ERO area is the averaging of the quantitative doppler and the PISA methods .However, this approach is time consuming.⁸

Invasive assessment

Mitral regurgitation could be assessed by invasive ventriculography. Some schools of thought have considered Left ventriculography as the reference methodology for assessment of mitral regurgitation. However this method of grading of MR is also subject to limitations and cannot thereby be considered gold standard because of influence of loading conditions affecting the MR severity.

Comparatively, Quantitative Doppler echocardiography is non invasive and also provides more objective data for grading and prognostic information about regurgitation than ventriculogram.⁵³

PROGNOSIS

The presence of ischaemic MR is an important indicator for long term morbidity as well as mortality. It is an independent risk factor for the same. The presence of ischaemic MR may result from acute infarction resulting in regional left ventricular dilation and consequent loss of contractile mechanism or in some instances it may be a previously existing lesion which went undiagnosed. Several studies have clearly shown that ischaemic mitral regurgitation can predict cardiovascular

mortality independently.^{2-4,8, 12, 54} Relative risk was found to be quite variable ranging from 1.48 - 7.5.⁸ Worse long term prognosis was seen in patients presenting with non ST elevation myocardial infarction.⁵⁵ A community based study has also confirmed the prognostic importance of ischaemic MR among one month survivors of MI: its presence is associated with a three-fold increase in the risk of heart failure and a 1.6-fold increased risk of death at 5-year follow-up, independent of LV ejection fraction, Killip class, age and gender.^{4,8} Barzilai et al found that AMI patients with a murmur suggestive of MR had a 12-month mortality of 36% compared with 15% for patients without an MR murmur.

⁵⁶SAVE study investigators also showed that mild MR was associated with high mortality. Greater the severity of mitral regurgitation, worse was the prognosis.⁴⁸ Even uncorrected mild MR, as well as moderate to severe ischaemic MR is found to be associated with higher mortality on long term. However, the severity of ischaemic MR tends to follow the severity of the LV dysfunction causing the MR; the worse the MR, the worse the LV functions. Till date, there are no studies to prove that ischaemic MR is a predictor of long term prognosis irrespective of severity of LV dysfunction present.⁸ No studies have proved that correction of mitral regurgitation will mitigate the long term mortality independent of left ventricular systolic dysfunction⁸

Though small case series has shown that coronary revascularisation in acute MI has reduced the severity of MR in selected patients, it tends to persist in > 50% of patients of MI on follow up.¹² Therefore, even coronary revascularisation may not halt its progression during long term.

Prognosis is also found to be related to the dynamicity of component of ischaemic mitral regurgitation. A 5 fold increase in the relative risk of death with an exercise-induced increase of ≥ 13 mm² of the ERO area was found.^{8, 57} It is the best predictor of hospitalisation and cardiovascular morbidity as compared to severity of mitral regurgitation at rest. Its deleterious effects are related to several factors like sudden increase in R Vol (regurgitant volume) and with rapid QRS widening due to increases in ventricular wall stress leading to worsening LV dyssynchrony.⁵⁸

In patients with LV systolic dysfunction, acute pulmonary oedema which may develop due to sudden worsening of dynamic MR leading to acute increase in left atrial pressures.^{8, 59} Greater exercise-induced regurgitant volume and systolic pulmonary arterial pressure may cause exertional dyspnea.⁶⁰ Around 20% may have an improvement in the severity of ischaemic mitral regurgitation during exercise, who have a favourable long term prognosis.⁵⁷ This is thought to happen in patients with contractile reserve in posterior segment.²⁹

MANAGEMENT

Numerous treatment options have been proposed but the treatment of ischaemic mitral regurgitation still remains a complex issue and needs further research and trials to find an ideal treatment modality with long term benefits. In current clinical approach at most places ring annuloplasty is preferred treatment modality.

However, long term benefits of this technique remains obscured as it has no role in correction of local alteration due to left ventricular remodelling.

Medical Management

Standard anti-failure medications such as angiotensin-converting enzyme (ACEI) inhibitor or ARBs if the ACEI is not well tolerated), aldosterone antagonist and beta-blockers .^{8, 61} It may help in alleviating the severity of mitral regurgitation by producing reverse left ventricular remodeling.^{8, 62}

Cardiac resynchronization therapy

Though biventricular pacing per se is not an treatment modality for ischaemic mitral regurgitation, it is an indication to consider CRT in patients of ischemic MR with reduced left ventricular ejection fraction ,functional class III or more even with medical treatment and ecg showing QRS of more than 120 ms.⁶³ Cardiac resynchronization therapy resynchronize the papillary muscles and increase the closing force which helps in immediate reduction in MR.^{8, 64-65} There is further

more reduction in the severity of MR in long term that is after few weeks or months as it plays a role in LV reverse remodelling, through a reduction in tethering .It can also reduce dynamic MR.⁶⁶ Magnitude of MR induced by exercise attenuate significantly in parallel to reverse Left Ventricular remodelling over a period of three months and result in improved cardiopulmonary performance.^{8, 67} Despite a reduction in the severity of MR, residual MR frequently persists. Immediate recurrence of MR has been seen post withdrawal of CRT due to dyssynchronization of the papillary muscles leads to.⁶⁸⁻⁷⁰

Percutaneous coronary revascularization

It may help in reducing the severity of mitral regurgitation at rest as well as during exertion in the subset where it is directly induced by ischaemia. .⁸

Surgical Management

Surgical approach for management of ischemic MR can only reduce its severity, not eliminate it completely. Coronary artery bypass grafting by itself is regarded insufficient in correction of MR.⁷¹ Persistence of even little residual mitral regurgitation postoperatively has been shown to be associated with higher mortality⁷² With the use of an undersized prosthetic (preferably two-sizes) ring⁷³, reduction in LV volume has been seen and even a small increase in LV ejection fraction has been documented ,⁷⁴ but the long-term benefits could not be

proved.^{75,76} Several studies have shown that long-term outcomes in terms of survival benefit or functional outcome is questionable by combined surgery.^{75,77-}

⁷⁸ In a recent randomized control trial it has been shown that mitral valve repair done along with CABG was associated with improvement of NYHA class, LVEF, and reduction in left ventricular diameter, left atrial size and PAP(pulmonary artery pressure).⁷⁹ It was not powered enough to analyse the effect on mortality. To conclude it may be said that fixing some valves may help, but it is difficult to identify which ones.⁸⁰ Recently published meta-analysis showed that mitral valve repair for ischaemic mitral regurgitation is associated with better survival compared with MVR (mitral valve replacement).⁸¹

The European Society of Cardiology guidelines recommend that patients with severe ischemic mitral regurgitation (ERO area ≥ 20 mm²) undergoing CABG should be treated by combined surgery (class I, level of evidence C).^{8,82} Mitral valve repair may be considered in symptomatic patients with severe mitral regurgitation who cannot be revascularized is questionable (class IIb).⁸² Mild mitral regurgitation should be managed conservatively. Due to the lacuna of well defined guidelines and evidence, the management of ischemic MR should be individualized. Assessment of myocardial viability, especially in region of posterior basal wall, inducible ischemia and the dynamic component of MR aids in decision making. Biphasic response or regional contractile reserve during stress

testing usually have exercise-induced reduction in mitral regurgitation, can be help decision making. Patients with exercise-induced increase in effective orifice area $\geq 13 \text{ mm}^2$ could be taken up for combined surgery. Severity of MR is always underestimated even with intra-operative TEE. Pharmacological measures like phenylephrine or rapid fluid challenge may be used to assess the ischaemic MR.⁸³ Parameters like grossly dilated left ventricle, multiple regurgitant jets, systolic sphericity index, wall motion score index, ESV(end-systolic volume), severe MR, $>2.5 \text{ cm}^2$ systolic tenting area, , large angle ($\geq 45^\circ$) of the posterior leaflet, $>1 \text{ cm}$ distance between coaptation point and mitral annulus are recognized as predictors of bad outcome of procedures like mitral valve repair by annuloplasty.⁸⁴⁻⁸⁶ Several adjunctive techniques have been proposed like chordal cutting, internal direct repositioning or external repositioning of the displaced papillary muscle.^{8, 87} However, they are not yet clinically approved for routine management of ischaemic mitral regurgitation.

PERCUTANEOUS REPAIR

Percutaneous edge-to-edge Alfieri procedure has been used for the treatment of MR due to either ischaemic or organic cause. In it, the central parts of both mitral leaflets are apposed producing a double orifice.^{8, 88} Many researchers have developed the devices which can be delivered in coronary sinus and reduce the

severity of mitral regurgitation by pushing the PML forward.⁸⁹⁻⁹⁰ Long term effects of these devices needs to be studied.

FORTHCOMING THERAPIES

Future targets like transplanting autologous myoblast has potential proven to cause localized LV reverse remodelling and appears to be a promising approach in decreasing ischaemic MR. More understanding of our concepts of leaflet adaptation in LV dysfunction can help us in developing potentially better therapies in future.⁹¹

3D EF Measurement in Myocardial Infarction

Echocardiography is the most common method to assess left ventricular systolic function. It can efficiently predict the outcome and help in determining the treatment modalities like CRT-D implantation etc. Routine 2D EF measurement has several limitations in AMI patients due to problems of foreshortening and geometric assumptions. 3D echo LVEF is much more accurate especially in presence of regional wall-motion abnormalities as it does not have geometric assumptions and is found to have comparable with present day “gold standard” cardiac MRI.⁽⁵⁾ It can automatically calculate ejection fraction and left ventricular mass using the automated softwares, and is therefore more reproducible. It is up to 3 times more accurate than 2DE LVEF.⁷

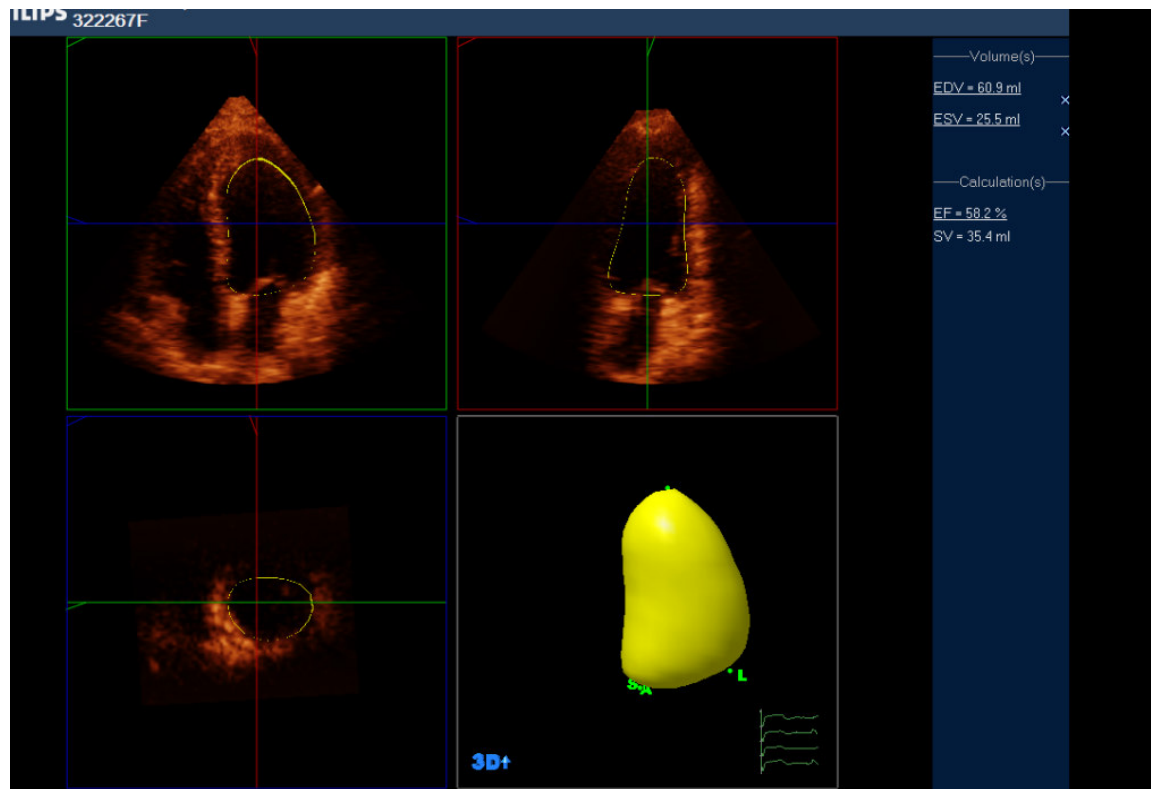


Figure 7.Measurement of 3D EF in echocardiography through Q lab analysis

DESIGN AND METHODOLOGY

Study Design:

This is a single centre prospective observational study done in the department of Cardiology, Christian Medical College (CMCH), Vellore.

SETTING

Study was done in the Cardiology department of Christian Medical College and Hospital, a tertiary care hospital in South India. 119 consecutive patients with STEMI were assessed for mitral regurgitation using various echocardiographic parameters.

STUDY PARTICIPITANTS

Inclusion criteria:

All patients older than 18 yrs old, who sustained STEMI between September 2011 to August 2012.

Exclusion criteria:

Patients were excluded from the study if they had

1. Rheumatic heart disease
2. Persistent arrhythmias
3. Known coronary artery disease(CAD)
4. Cardiogenic shock

5. Trivial MR

Methods:

1. Demographic and clinical profile of patients were collected were collected once they consented
2. All patients underwent echo once they were stable during index admission. Mitral regurgitation quantified using jet width[vena contracta], jet area and proximal isovelocity surface area(PISA) ,if central jet is present
3. Left ventricular ejection fraction were assessed by both Simpson's method as well as 3D echocardiography

STATISTICAL ANALYSIS

Statistical analysis was done using commercially available statistical software ('IBM SPSS software version 15', Illinois, Chicago). All continuous variables are expressed as mean \pm SD and categorical variables are expressed as number (percentages). Independent samples T test was used for comparative analysis of two groups with a normally distributed continuous variables. Comparison of categorical variables was done using Chi square test. Pearson correlation coefficient was used to analyze the correlation between two continuous variables with a normal distribution. Comparative

analysis was performed by one way analysis of variance (ANOVA). A p value less than 0.05 was considered statistically significant for all test results.

RESULTS

1. STUDY PROFILE AND BASELINE CHARACTERISTICS

A total of 119 patients of STEMI were evaluated for ischaemic MR during the study period. Mean age of patients in the whole study population was 53.42 ± 11.47 years. Majority patients (57.1%) were found to be in the age group of 40-59 years, with least number was in >80 yrs (2.5%). 10.9% patients were young, <40 years having MI. Number of males outnumber within each group. Ratio of Males: Female is 96:23. 31.9%(38) of the patients has type 2 diabetes mellitus, 98.3%(117) of patients had dyslipidemia, 37.8(45) % of the patients had hypertension, 54.6% (65) were obese, 47.1%(56) were current smoker and 13.4%(16) had positive family history of coronary artery disease(CAD). On an average all patients were had at least 2 risk factors.

Baseline characteristics of both groups with Anterior wall MI (AWMI) and Inferior wall MI (IWMI) were comparable without any significant statistical difference except baseline 2D EF, which was higher in IWMI group (table 1). Patients presenting with AWMI were having slightly higher proportion of hypertension, though it was statistically insignificant.

Characteristic	AWMI	IWMI	p value
Age	52.30±11.11	54.58±11.81	.281
Male	47(78.3%)	49(83.1%)	.515
Female	13(21.7%)	10(16.9%)	.515
Diabetes	19(31.7%)	19(32.2%)	.85
Hypertension	27(45%)	18(30.5%)	.103
Smoking	29(48.3%)	27(45.8%)	.779
Dyslipidemia	59(98.3%)	58(98.3%)	.990
Obesity	35(58.3%)	30(53.1%)	.293
Positive Family History	10(16.7%)	6(10.2%)	.299
Baseline 2D EF	44.94±7.5	50.7±7.4	.000(significant)
Baseline 3D EF	45.42±8.5	46.96±8.6	.33

Table 1. Baseline characteristics of patients according to the type of STEMI

2. Prevalence of MR

27(22.7%) patients were found to have MR during index admission for MI (Figure 8), out of which 24(88.9%) had mild MR and 3(11.1%) had moderate MR based on quantitative measurements. None of the patients were found to have severe MR.

Mean Vena contracta (VC) in mild and moderate MR patients at presentation were 2.91 ± 0.94 and 3.54 ± 0.25 respectively. Mean PISA radius, effective regurgitant orifice area (ERO) and regurgitation volume in mild MR patients at presentation were 0.315 ± 0.08 , 0.05 ± 0.03 and 7.07 ± 3.65 respectively (table 2).

MR	VC(mm)	PISA radius(cm)	ERO(cm ²)	MR Volume(ml)
MILD(n=24)	2.91 ± 0.94	0.315 ± 0.08	0.05 ± 0.03	7.07 ± 3.65
MODERATE(n=3)	3.54 ± 0.25	0.44 ± 0.06	0.10 ± 0.06	30.33 ± 7.77
AFTER 1 MONTH				
MILD(n=12)	2.97 ± 0.55	0.38 ± 0.81	0.05 ± 0.03	6.9 ± 3.17
MODERATE(n=2)	4.8 ± 8.5	0.55 ± 0.07	0.125 ± 0.02	27 ± 1.41

Table 2. Quantitative echocardiographic parameters of MR at presentation and after 1 month

Mean age of patients presenting with ischaemic MR at onset was 59.2 ± 10.82 (figure 9), population includes 74.1% males (20) and 25.9 % (7) females. Majority of patients presenting with MR at admission were in the age group of 60-79 years (figure 9).Among the patients having MR at admission included 48.1%(13) type 2 diabetics, 100%(27) dyslipidemics, 33.3 % (9) hypertensive's, 44.4% (12) obese, 44.4 %(12) current smoker, 22.2%(6) had AWTMI, 77.8%(21) had IWTMI and none had a positive family history of coronary artery disease (CAD). Though there was trend towards females being more commonly associated with MR, it was found to be statistically insignificant (figure 10). None of the risk factors predicted of onset of MR in STEMI patients at admission and during follow up on statistical analysis except age, diabetes and site of STEMI(Table 3).IWTMI patients were more likely to develop MR at presentation (table 3).Male gender was found to have higher prevalence of absence of MR during follow up.

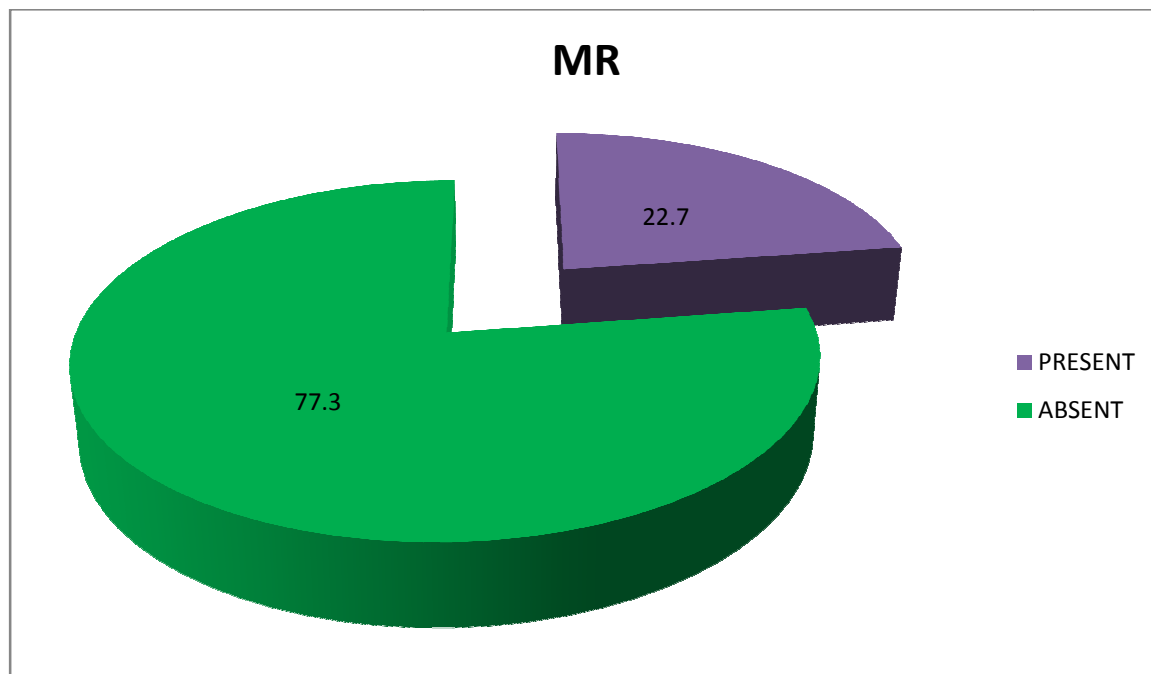


Figure 8.Prevalence of MR at admission

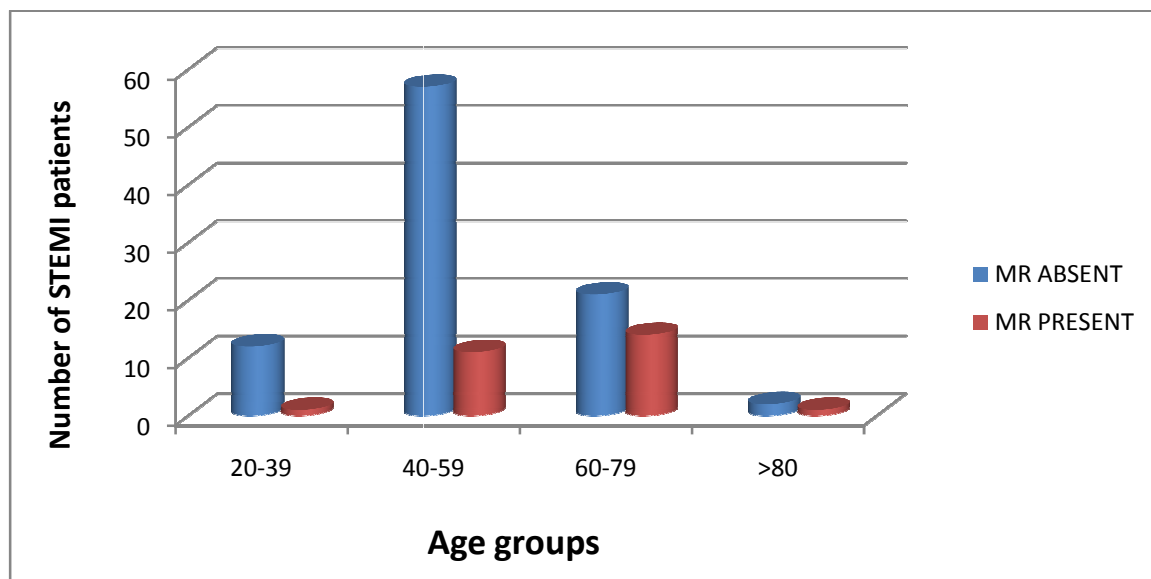


Figure 9.Prevalence of MR according to the age groups

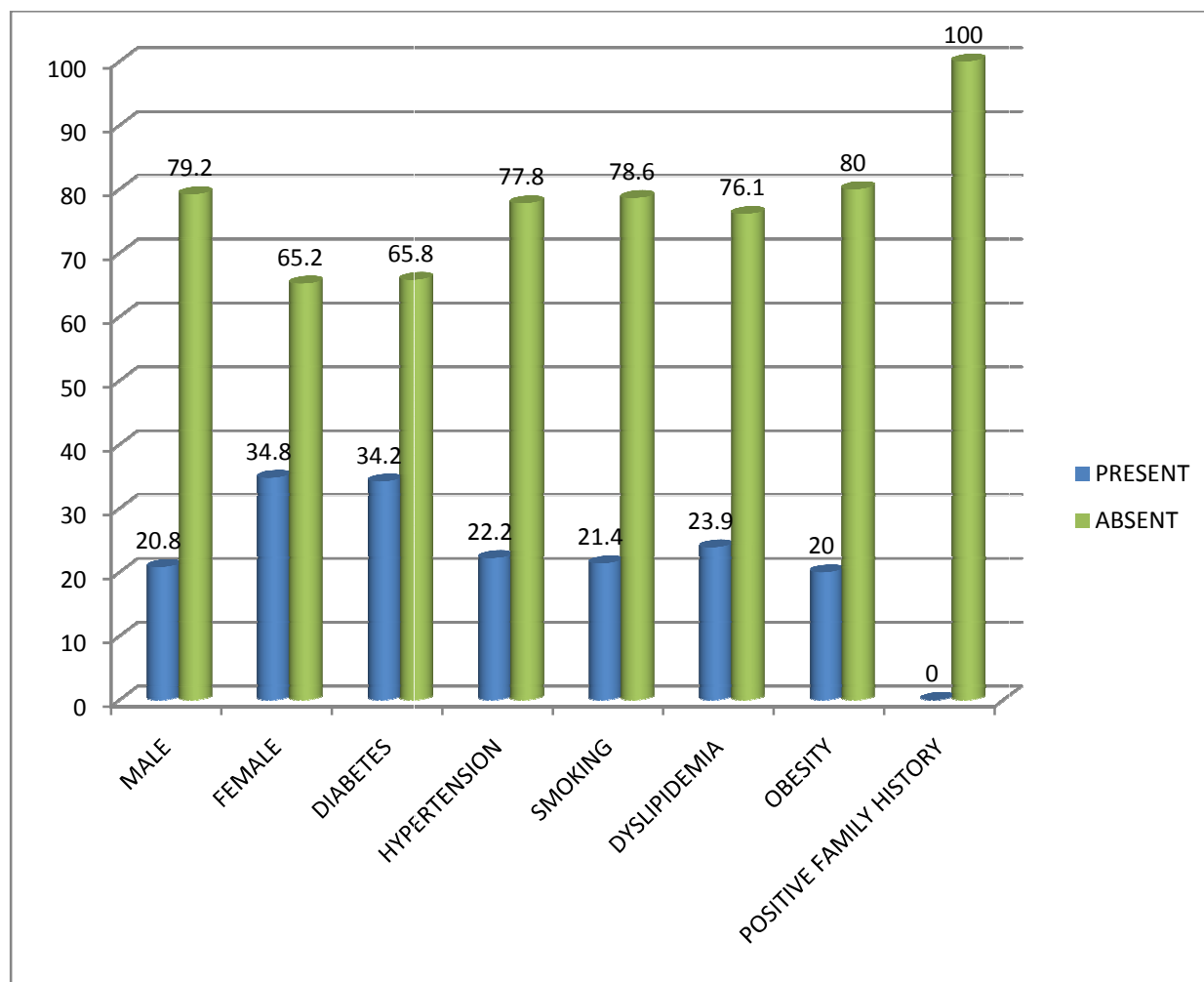


Figure 10. Proportion of baseline risk factors of patients with and without MR

During follow up after 1 month, MR was found to be present in 15.7% of patients despite a dropout of 30(25.2%) patients (figure 11). MR continue to persist in 10.1%(9) patients with improvement in severity in 13.5 %(12) and new onset in 5.6 %(5) patients (figure 12). Among 14 patients who were having MR at follow up, 3(21.4%) had AWMi and 11(78.6%) had IWMi at baseline. None of the baseline characteristic was found to be predictor of mitral regurgitation during follow up

(table 3) excluding sex and site of STEMI. AWMIs and males tend to have lesser prevalence of mitral regurgitation at follow up (table 4). IWMI patients continue to have higher incidence of Mitral Regurgitation which was persistent and also developed new onset MR, but it was not statistically significant ($p=0.647$). Females have higher prevalence of Mitral Regurgitation during follow up visit (table 3). None of the patients with positive family history had Mitral Regurgitation at onset. Development of MR in STEMI patients was not influenced by the treatment modality used (figure 13).

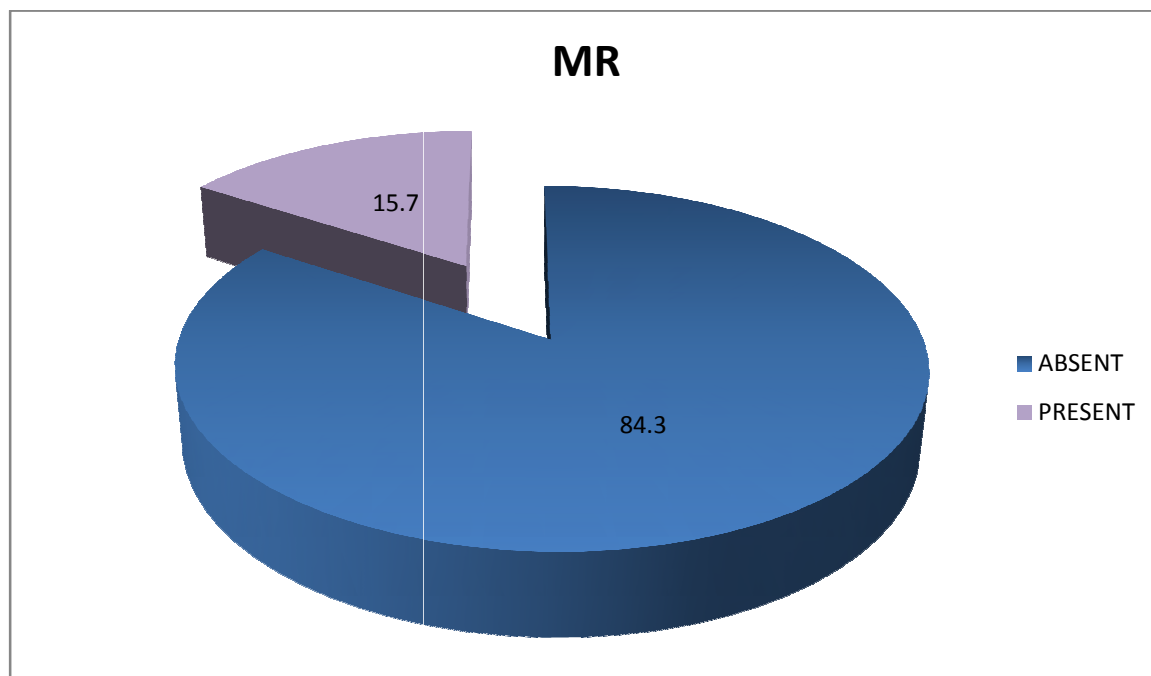


Figure 11. Prevalence of MR after 1 month

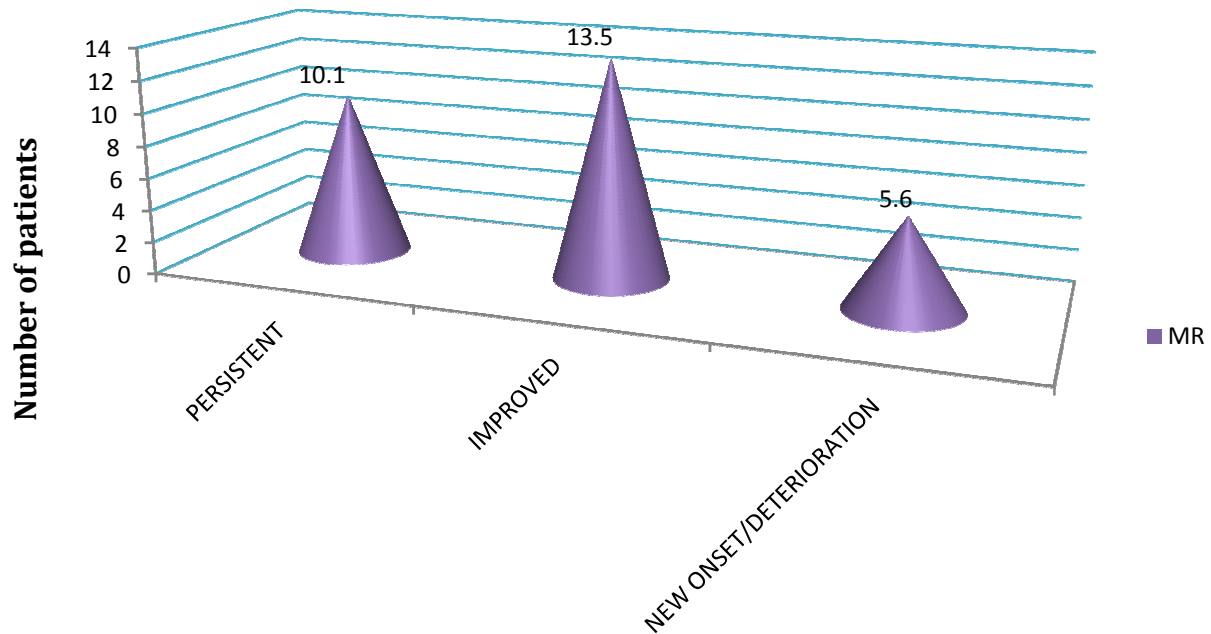


Figure 12. Profile of MR after 1 month

Proportion of patients having greater severity of MR after 1 month was found to be slightly higher, despite being reduction in absolute number of patients having moderate MR (figure 14). Initially, 3(11.1%) patients had moderate MR, but at 1 month, only 2 patients had moderate MR. IWMI patients were found to have higher prevalence of MR (figure 15).

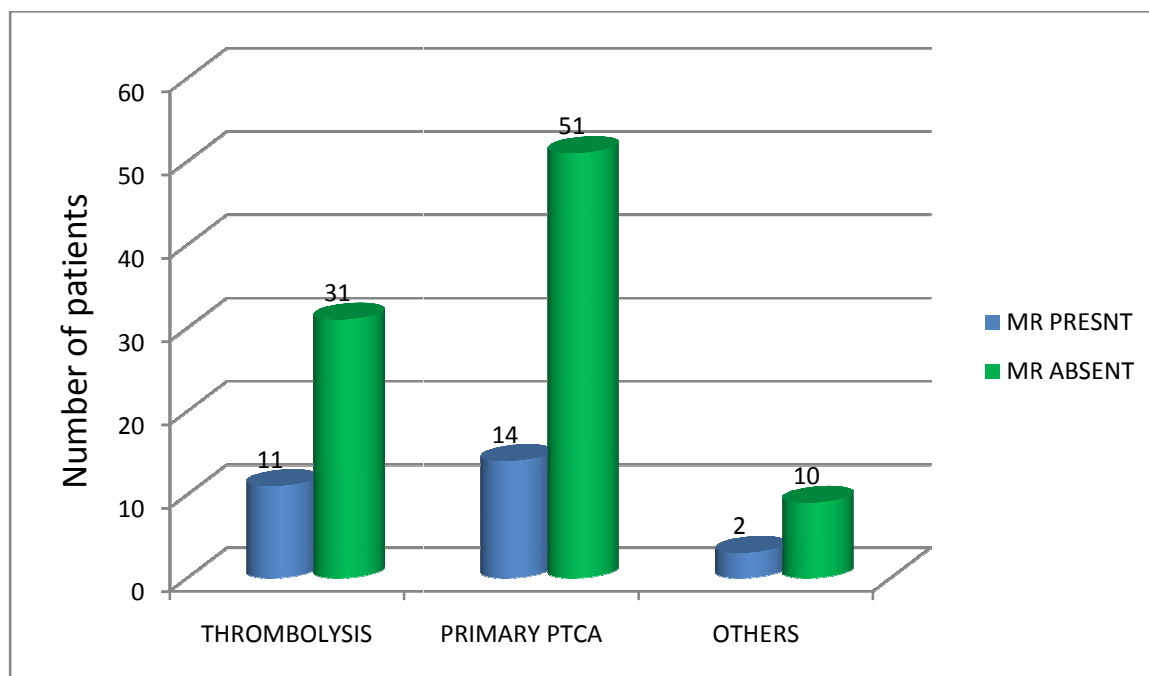


Figure 13. Prevalence of MR in relation to treatment strategies

Characteristic	Baseline MR	p value	MR at 1 month	p value
Age (yrs)				
Group 1 (20-39)	1(3.7)	0.024*	2(14.3)	0.132
Group 2 (40-59)	11(40.7)		5(35.7)	
Group 3 (60-79)	14(51.9)		7(50)	
Group 4 (≥ 80)	1(3.7)		0(0)	
Sex				
Male(96)	20(74.1)	0.323	9(64.3)	0.007*
Female(23)	7(25.9)		5(35.7)	

Site of STEMI				
AWMI(60)	6(22.2)	0.001*	3(21.4)	0.03*
IWMI(59)	21(77.8)		11(78.6)	
Diabetes(n=38)	13(48.1)	0.04*	5(35.7)	0.612
Hypertension(n=45)	9(33.3)	0.585	4(28.6)	0.615
Smoking(n=56)	12(44.4)	0.757	5(35.7)	0.210
Dyslipidemia(n=117)	27((100)	0.44	14(100)	0.539
Obesity(n=65)	12(44.4)	0.36	5(35.7)	0.257
Positive Family History(n=16)	0(0)	0.02*	1(7.1)	0.458
Baseline 2D EF	48.17±8.93	0.769	50.2±8.6	0.216
Baseline 3D EF	46.24±7.68		50.37±8.61	
Reperfusion modality				
Thrombolysis (n=45)	11(40.7)	0.744	8(57.1)	0.134
Primary PCI (n=62)	14(51.9)		6(42.9)	
Miscc(n=12)	2(7.4)		0(0)	

Table 3.Comparison of various variables with MR at onset and during follow up

*statistically significant

MR		Sex		Total	p value
		1(Male)	2(Female)		
Absent	Number of patients	69	7	76	0.019*
		(88.5%)	(58.3%)	84.4%	
Present	Number of patients	9	5	14	
		(11.5%)	(41.7%)	15.6%	
Total	Total no.	78	12	90	
	% within sex	100.0%	100.0%	100.0%	

Table 4. Association of sex with the prevalence of Mitral Regurgitation at 1 month

Severity	Baseline	MR at FU
MR		
Mild	24(88.9)	12(85.7)
Moderate	3(11.1)	2(14.2)
Severe	-	-

Figure 14. Prevalence of severity of MR at onset and after 1 month

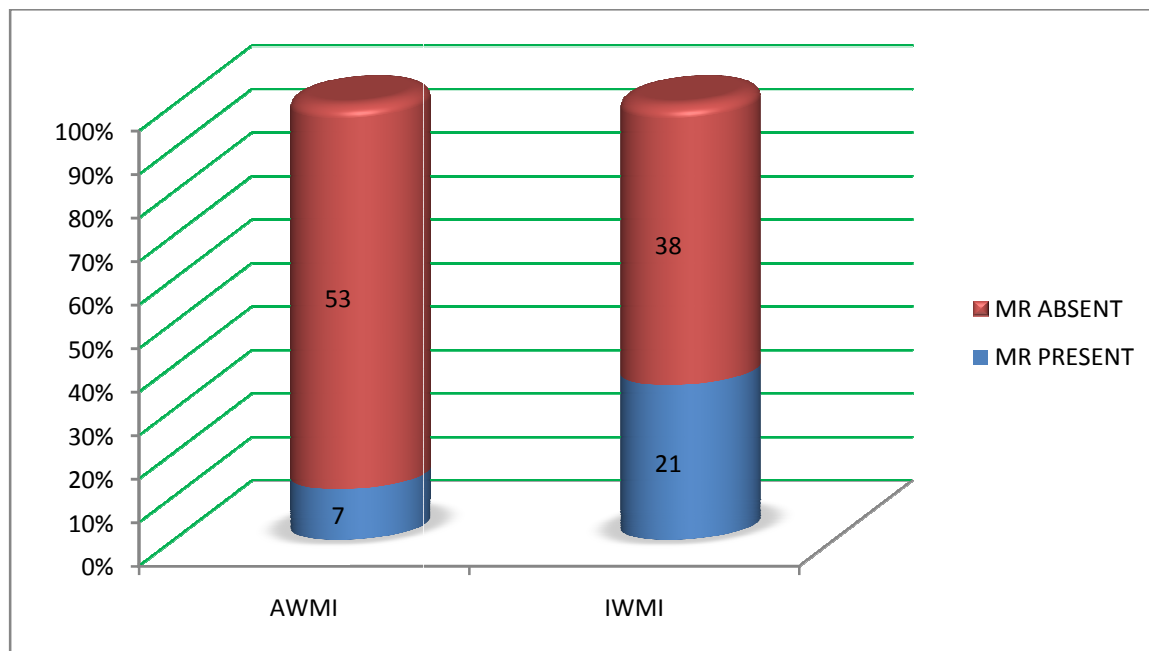


Figure 15. Prevalence of MR in relation to type of MI

3. Correlation among 2D and 3D EF in STEMI

Mean 2D EF in AWMI group at presentation was $44.94\% \pm 7.5\%$ and in IWMI group was slightly higher $50.7\% \pm 7.4.4\%$. LV systolic functions was found to be better in IWMI patients on presentation. After 1 month, there was an increase in 2D EF by 6.9% (51.83 ± 8.06) in AWMI group and negligible change in IWMI group (51.05 ± 8.4). 2D EF was found to strongly correlate with 3D EF at admission and during follow up irrespective of type of STEMI (table 5), though poor image quality was one of the hindering factor in some of the patients. Figure 14 shows the scatter plot displaying the distribution of 2D and 3D EF at baseline.

2D and 3D EF	Correlation coefficient, r	p (2 tailed)
At admission	0.525	0.01
At 1 month	0.609	0.01

Table 5. Correlations of 2D and 3D EF at admission and after 1 month

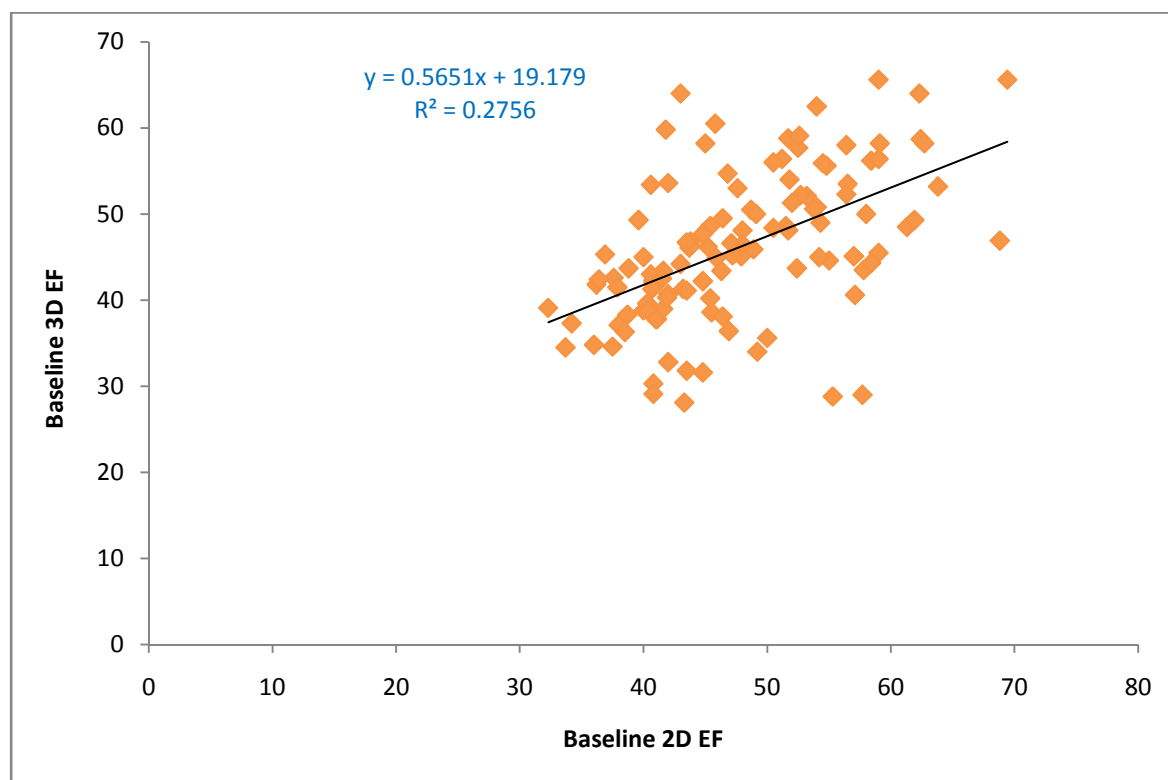


Figure 16. Scatter plot showing relationship of 2D-EF and 3D-EF at admission (Pearson correlation coefficient r, 0.525)

* Correlation is significant at the 0.01 level (2-tailed).

DISCUSSION

The present study is a prospective observational study which evaluated the prevalence of functional (ischaemic) mitral regurgitation in patients with STEMI at presentation and during 1 month of follow up. Mean age of patients was 53.42 ± 11.47 years with majority of them being males (83%). Most common age group was found to be 40-59 years (57.1%), with least patients in >80 yrs (2.5%). 10.9% patients had MI in young (<40 years). Our results are found to be similar as observed in a large retrospective analysis by Brijesh et al⁹² done in our institute. Most common risk factor was found to be dyslipidemia (98.3%), followed by obesity (54.6%), smoking (47.1%), diabetes (31.9%), hypertension (37.8%) and positive family history of coronary artery disease (13.4%). This data is different from create study where diabetics 34% had diabetes; 37.7% had hypertension; and 40.2% were smoker.⁹³ All patients were having multiple coronary risk factors. LV systolic function was found to be better in IWMI patients at presentation as lesser territory was compromised by ischaemia. Similar results were observed by Darbar et al and our findings are consistent with it.⁹⁴ With anterior infarction, the injury is exclusively in the left ventricle, whereas inferior infarction is associated with injury to both ventricles, causing less impairment of left ventricular function despite an equivalent overall myocardial insult. After 1 month, there was a trend in the increase in 2D EF by 6.9% in AWMi group due to revascularisation therapy.⁹⁵

MR was found in 22.7% at presentation of STEMI in our study population, which is in accordance with older studies including angiographic ones (table 5). It was found to be higher in older age group, diabetics and IWMI patients at the onset, which was consistent with previous studies.^{2, 12, 15} There was a trend toward an excess of women in the MR group, though it was not statistically significant due to under representation of females in present study. Similar data on the demographics of MR after AMI have been presented earlier by Lehmann, Tchong, and Barzilai.^{2, 12, 15} IWMI involves the posterior wall of LV leading to left ventricle remodelling and distortion. This result in apico-lateral as well as posterior displacement of papillary muscle, which in turn leads to apical displacement of mitral leaflet coaptation point, producing the ischaemic mitral regurgitation.⁸ Mostly patients had mild MR and none had severe MR which is possibly due to exclusion of very sick patients like cardiogenic shock. During follow up after 1 month, MR was found to be in 15.7% of patients despite a dropout of 30(25.2%) patients. Some patients with MR tend to improve with the resolution of acute ischaemia due to revascularisation as well as favourable LV remodelling and improvement in LV systolic function, while some had deteriorated with worsening mitral regurgitation due to adverse left ventricular remodelling with passage of time after STEMI. Treatment modality used didn't influence the development of MR as shown earlier

by Tcheng JE et al. Acute reperfusion with thrombolysis or angioplasty did not reliably reverse valvular incompetence as seen in earlier observational studies.

None of the baseline risk factor except diabetes, territory involved and older age group ,can reliably predict the development of MR at onset and left ventricle systolic function, though this study was not powered enough to study those predictors. IWMi patients tend to have higher prevalence of MR during the STEMI as well as during follow up secondary to tethering effect on the mitral valve leaflet which may be a harbinger of chronic MR later on. Treatment modality didn't influence the severity of MR in STEMI patients.

Author	Year	No of patients	Modality used	Prevalence of MR (%)
Barzilai B et al¹⁵	1988	1480	Echo	39
Tcheng JE et al¹²	1992	1480	Echo	17.9
Lehmann KG et al²	1992	206	LV ventriculography	13
Gervasio A. Lamas et al³	1997	727	LV ventriculography	19.4
Present study	2013	119	Echo	22.7

2D EF was found to correlate linearly with 3D EF irrespective of type of STEMI.

In a recently published metanalysis, Jennifer L. Dorosz et al⁹⁶ showed similar results. With EF, there is no difference in the bias between 3DE and 2DE, and the difference in the variance is modest ($\pm 4.7\%$). Despite being subjected to errors due to foreshortening, poor endocardial definition, narrow echocardiographic windows, and assumptions about LV shape, it remains a ubiquitous tool for assessing LV size and systolic function in day to day clinical practise.

Although 3DE shows promise in providing the accessibility of echocardiography and the multi- planar imaging of CMR, this nascent technology still has limited spatial and temporal resolution compared with gold standard modality of CMR. Despite its limitations, 3DE may be superior to 2D techniques.

Our single centre study shades light on the prevalence of Mitral Regurgitation in acute STEMI in Indian population. MI is most prevalent in middle age persons with ischaemic Mitral Regurgitation in slightly older age group. Modalities of revascularisation (mechanical vs thrombolytic or medical treatment doesn't influence the course of Mitral Regurgitation in short term follow up. However, these findings need to be confirmed in larger study population MI.

STUDY LIMITATIONS

The main limitation of this study was smaller sample size, which was not powered enough to study the predictors of development of MR in STEMI patients.

Moreover, females were underrepresented in the study group, which may have negated the gender effect on ischemic MR. Critically ill patients , cardiogenic shock etc were not included in whom ,there is a probability of having severe mechanical complications of STEMI like severe MR. There was a significant drop out during follow up, which may have influenced the statistical analysis.

Only echocardiographic LV systolic function measurements were used and were not compared with other imaging modalities like MRI, which is gold standard for determining EF in patients with regional wall motion abnormalities. Longer follow up periods are required for assessing the development of chronic ischaemic MR.

CONCLUSION

STEMI patients have 22.7% prevalence of MR at onset, which is more likely in older age group, diabetics and IWMI patients and independent of gender and left ventricle systolic dysfunction. Despite under-representation of females in our study population, they seem to carry higher risk of development of ischaemic Mitral Regurgitation in India. Most commonly, ischaemic MR is of milder severity at onset which tends to persist in 15.7% of patients during follow up. There is a significant association of development of ischaemic MR during acute STEMI with IWMI. Some patients may develop new MR later on depending on the LV remodelling. 2D EF estimation with a properly acquired good image is comparable to more precise 3D EF in STEMI patients.

BIBLIOGRAPHY

1. Messika-Zeitoun D, Fung Yiu S, Grigioni F, Enriquez-Sarano M. Role of Echocardiography in the Detection and Prognosis of Ischemic Mitral Regurgitation. *Rev Esp Cardiol* 2003;56(6):529-34.
2. Lehmann KG, Francis CK, Dodge HT. Mitral regurgitation in early myocardial infarction. Incidence, clinical detection, and prognostic implications. TIMI Study Group. *Ann. Intern. Med.* 1992; 117(1):10–7.
3. Lamas GA, Mitchell GF, Flaker GC, Smith SC Jr, Gersh BJ, Basta L, et al. Clinical significance of mitral regurgitation after acute myocardial infarction. Survival and Ventricular Enlargement Investigators. *Circulation*. 1997; 96(3):827–33.
4. Bursi F, Enriquez-Sarano M, Nkomo VT, Jacobsen ST, Westo SA, Meverden RA, Roger VL. Heart failure and death after myocardial infarction in the community: the emerging role of mitral regurgitation. *Circulation* 2005; 111:295–301.
5. McCully RB, Enriquez-Sarano M, Tajik AJ, Seward JB. Overestimation of severity of ischemic/functional mitral regurgitation by color Doppler jet area. *Am. J. Cardiol.* 1994; 74(8):790–3.

6. Lancellotti P, Moura L, Pierard LA, Agricola E, Popescu BA, Tribouilloy C et al. European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 2: mitral and tricuspid regurgitation (native valve disease). *European Journal of Echocardiography* 2010; 11:307–332.
7. Nikitin NP, Constantin C, Loh PH, Ghosh J, Lukaschuk EI, Bennett A, et al. New generation 3-dimensional echocardiography for left ventricular volumetric and functional measurements: comparison with cardiac magnetic resonance. *Eur J Echocardiogr.* 2006; 7(5):365–72.
8. Piérard LA, Carabello BA. Ischaemic mitral regurgitation: pathophysiology, outcomes and the conundrum of treatment. *Eur Heart J.* 2010 Dec; 31(24):2996-3005.
9. Levine RA, Schwammenthal E. Ischemic mitral regurgitation on the threshold of a solution: from paradoxes to unifying concepts. *Circulation* 2005; 112:745–758.
10. Van Mieghem NM, Piazza N, Anderson RH, Tzikas A, Nieman K, De Laet LE et al. Anatomy of the mitral valvular complex and its implications for transcatheter interventions for mitral regurgitation. *J Am Coll Cardiol.* 2010; 56(8):617-26.
11. Chaput M, Handschumacher MD, Tournoux F, Hua L, Guerrero JL, Vlahakes GJ, Levine RA. Mitral leaflet adaptation to ventricular remodelling occurrence and

adequacy in patients with functional mitral regurgitation. *Circulation* 2008; 118:845–852.

12. Tcheng JE, Jackman JD, Nelson CL, et al. Outcome of patients sustaining acute ischemic mitral regurgitation during myocardial infarction. *Ann Intern Med* 1992; 117:18–24.

13. Mittal AK, Langston M, Cohn KE, Selzer A, Kerth W. Combined papillary muscle and left ventricular wall dysfunction as a cause of mitral regurgitation: an experimental study. *Circulation*. 1971; 44:174-180.

14. Kono T, Sabbah HN, Rosman H, Alam M, Jafri S, Stein PD, Goldstein S. Mechanism of functional mitral regurgitation during acute myocardial ischemia. *J Am Coll Cardiol*. 1992; 19:1101-1105.

15. Barzilai B, Gessler C, Pérez JE, Schaab C, Jaffe AS. Significance of Doppler-detected mitral regurgitation in acute myocardial infarction. *Am J Cardiol*. 1988; 61:220-223.

16. McCarthy KP, Ring L, Rana BS. Anatomy of the mitral valve: understanding the mitral valve complex in mitral regurgitation. *Eur J Echocardiogr*. 2010;11(10):i3-9.

17. Carpentier A. Cardiac valve surgery — the “French correction.” *J Thorac Cardiovasc Surg.* 1983; 86:323-37.
18. Yiu S, Enriquez-Sarano M, Tribouilloy C, Seward J, Tajik A. Determination of the degree of functional mitral regurgitation in patients with systolic left ventricular dysfunction: a quantitative clinical study. *Circulation* 2000; 102:1400–1406.
19. Messas E, Guerrero JL, Handschumacher MD, Conrad C, Chow CM, Sullivan S et al. Chordal cutting: a new therapeutic approach for ischemic mitral regurgitation. *Circulation* 2001; 104:1958–1963.
20. Watanabe N, Ogasawara Y, Yamaura Y, Kawamoto T, Toyota E, Akasaka T, Yoshida K. Quantitation of mitral valve tenting in ischemic mitral regurgitation by transthoracic real-time three-dimensional echocardiography. *J Am Coll Cardiol* 2005; 45:763–769.
21. Bursi F, Enriquez-Sarano M, Jacobsen SJ, Roger VL. Mitral regurgitation after myocardial infarction: a review. *Am J Med* 2006; 119:103–112.
22. Carabello BA. Ischemic mitral regurgitation and ventricular remodelling. *J Am Coll Cardiol* 2004; 43:384–385.

23. Yoran C, Yellin EL, Becker RM, Gabbay S, Frater RW, Sonnenblick EH. Dynamic aspects of acute regurgitation: effects of ventricular volume, pressure and contractility on the effective regurgitant orifice area. *Circulation* 1979; 60:170–176.
24. Olson L, Subramanian R, Ackermann D, Orszulak T, Edwards W. Surgical pathology of the mitral valve: a study of 712 cases spanning 21 years. *Mayo Clin Proc* 1987; 62:22–24.
25. Yellin E, Yoran C, Sonnenblick E, Gabbay S, Frater R. Dynamic changes in the canine mitral regurgitant orifice area during ventricular ejection. *Circ Res* 1979; 45:677–683.
26. Schwammenthal E, Chen C, Benning F, Block F, Breithardt G, Levine R. Dynamics of mitral regurgitant flow and orifice area. Physiologic application of the proximal flow convergence method: clinical data and experimental testing. *Circulation* 1994; 90:307–322.
27. Rosario LB, Stevenson LW, Solomon SD, Lee RT, Reimold SC. The mechanism of decrease in dynamic mitral regurgitation during heart failure treatment: importance of reduction in the regurgitant orifice size. *J Am Coll Cardiol* 1998; 32: 1819–1824.

28. Lebrun F, Lancellotti P, Pie´rard LA. Quantitation of functional mitral regurgitation during bicycle exercise in patients with heart failure. *J Am Coll Cardiol* 2001; 38: 1685–1692.
29. Lancellotti P, Lebrun F, Pie´rard LA. Determinants of exercise-induced changes in mitral regurgitation in patients with coronary artery disease and left ventricular dysfunction. *J Am Coll Cardiol* 2003; 42:1921–1928.
30. Lancellotti P, Pie´rard LA. Chronic ischaemic mitral regurgitation: exercise testing reveals its dynamic component. *Eur Heart J* 2005; 26:1816–1817.
31. Desjardin VA, Enriquez-Sarano M, Tajik AJ, Bailey KR, Seward JB. Intensity of murmurs correlates with severity of valvular regurgitation. *Eur Heart J* 1996; 17:149–156.
32. Chaliki HP, Nishimura RA, Enriquez-Sarano M, Reeder GS. A simplified, practical approach to assessment of severity of mitral regurgitation by Doppler color flow imaging with proximal convergence: validation with concomitant cardiac catheterization. *Mayo Clin Proc* 1998; 73:929–35.
33. Enríquez-Sarano M, Tajik A, Bailey K, Seward J. Color flow imaging compared with quantitative Doppler assessment of severity of mitral regurgitation: Influence of eccentricity of jet and mechanism of regurgitation. *J Am Coll Cardiol* 1993; 21:1211-9.

34. Tribouilloy C, Shen WF, Quére´ JP, Rey JL, Choquet D, Dufosse´ H et al. Assessment of severity of mitral regurgitation by measuring regurgitant jet width at its origin with transesophageal Doppler color flow imaging. *Circulation* 1992; 85:1248–53.
35. Heinle SK, Hall SA, Brickner ME, Willett DL, Grayburn PA. Comparison of vena contracta width by multiplane transesophageal echocardiography with quantitative Doppler assessment of mitral regurgitation. *Am J Cardiol* 1998; 81:175–9.
36. Hall SA, Brickner ME, Willett DL, Irani WN, Afridi I, Grayburn PA. Assessment of mitral regurgitation severity by Doppler color flow mapping of the vena contracta. *Circulation* 1997; 95:636–42.
37. Matsumura Y, Fukuda S, Tran H, Greenberg NL, Agler DA, Wada N et al. Geometry of the proximal isovelocity surface area in mitral regurgitation by 3-dimensional color Doppler echocardiography: difference between functional mitral regurgitation and prolapse regurgitation. *Am Heart J* 2008;155:231–8.
38. Song JM, Kim MJ, Kim YJ, Kang SH, Kim JJ, Kang DH et al. Three-dimensional characteristics of functional mitral regurgitation in patients with severe left ventricular dysfunction: a real-time three-dimensional colour Doppler echocardiography study. *Heart* 2008; 94:590–6.

39. Yosefy C, Hung J, Chua S, Vaturi M, Ton-Nu TT, Handschumacher MD et al.

Direct measurement of vena contracta area by real-time 3-dimensional echocardiography for assessing severity of mitral regurgitation. *Am J Cardiol* 2009; 104: 978–83.

40. Kahlert P, Plicht B, Schenk IM, Janosi RA, Erbel R, Buck T. Direct assessment of size and shape of noncircular vena contracta area in functional versus organic mitral regurgitation using real-time three-dimensional echocardiography. *J Am Soc Echocardiogr* 2008; 21:912–21.

41. Khanna D, Vengala S, Miller AP, Nanda NC, Lloyd SG, Ahmed S et al. Quantification of mitral regurgitation by live three-dimensional transthoracic echocardiographic measurements of vena contracta area. *Echocardiography* 2004; 21:737–43.

42. Messika-Zeitoun D, Bellamy M, Avierinos JF, Breen J, Eusemann C, Rossi A et al. Left atrial remodelling in mitral regurgitation--methodologic approach, physiological determinants, and outcome implications: a prospective quantitative Doppler-echocardiographic and electron beam-computed tomographic study. *Eur Heart J*. 2007 Jul; 28(14):1773-81.

43. Enriquez-Sarano M, Bailey KR, Seward JB, Tajik AJ, Krohn MJ, Mays JM. Quantitative Doppler assessment of valvular regurgitation. *Circulation* 1993; 87:841–848.
44. Enriquez-Sarano M, Avierinos JF, Messika-Zeitoun D, Detaint D, Capps M, Nkomo V et al. Quantitative determinants of the outcome of asymptomatic mitral regurgitation. *N Engl J Med* 2005; 352:875–83.
45. Enriquez-Sarano M, Seward JB, Bailey KR, Tajik AJ. Effective regurgitant orifice area: a non-invasive Doppler development of an old hemodynamic concept. *J Am Coll Cardiol* 1994; 23:443–451.
46. Enríquez-Sarano M, Miller FJ, Hayes S, Bailey K, Tajik A, Seward J. Effective mitral regurgitant orifice area: clinical use and pitfalls of the proximal isovelocity surface area method. *J Am Coll Cardiol* 1995; 25:703-9.
47. Lancellotti P, Troisfontaines P, Toussaint AC, Pie´rard LA. Prognostic importance of exercise-induced changes in mitral regurgitation in patients with chronic ischemic left ventricular dysfunction. *Circulation* 2003; 108:1713–7.
48. Grigioni F, Enriquez-Sarano M, Zehr KJ, Bailey KR, Tajik AJ. Ischemic mitral regurgitation: long-term outcome and prognostic implications with quantitative Doppler assessment. *Circulation* 2001; 103:1759–1764.

49. Vandervoort P, Rivera J, Mele D, Palacios I, Dinsmore R, Weyman A, et al. Application of color Doppler flow mapping to calculate effective regurgitant orifice area. An in vitro study and initial clinical observations. *Circulation* 1993; 88:1150-6.
50. Schwammenthal E, Chen C, Benning F, Block F, Breithardt G, Levine R. Dynamics of mitral regurgitant flow and orifice area. Physiologic application of the proximal flow convergence method: clinical data and experimental testing. *Circulation* 1994; 90:307–322.
51. Yosefy C, Levine RA, Solis J, Vaturi M, Handschumacher MD, Hung J. Proximal flow convergence region as assessed by real-time 3-dimensional echocardiography: challenging the hemispheric assumption. *J Am Soc Echocardiogr* 2007; 20: 389–396.
52. Iwakura K, Ito H, Kawano S, Okamura A, Kurotobi T, Date M et al. Comparison of orifice area by transthoracic three-dimensional Doppler echocardiography versus proximal isovelocity surface area (PISA) method for assessment of mitral regurgitation. *Am J Cardiol* 2006; 97:1630-7.
53. Lung B. Management of ischaemic mitral regurgitation. *Heart* 2003; 89(4):459-64.

54. Feinberg MS, Schwammenthal E, Shlizerman L, Porter A, Hod H, Freimark D et al. Prognostic significance of mild mitral regurgitation by color Doppler echocardiography in acute myocardial infarction. *Am J Cardiol* 2000; 86:903–907.
55. Perez de Isla L, Zamorano J, Quezada M, Almeria C, Rodrigo JL, Serra V, Rubira JCG, Ortiz AF, Macaya C. Prognostic significance of functional mitral regurgitation after a first non-ST-segment elevation acute coronary syndrome. *Eur Heart J* 2006; 27:2655–2660.
56. Barzilai B, Davis VG, Stone PH, Jaffe AS. Prognostic significance of mitral regurgitation in acute myocardial infarction. *Am J Cardiol* 1990; 65:1169-1175.
57. Lancellotti P, Ge´rard PL, Pie´rard LA. Long-term outcome of patients with heart failure and dynamic functional mitral regurgitation. *Eur Heart J* 2005;26: 1528–1532.
58. Lancellotti P, Kulbertus HE, Pie´rard LA. Predictors of rapid QRS widening in patients with coronary artery disease and left ventricular dysfunction. *Am J Cardiol* 2004; 93:1410–1412.
59. Pie´rard LA, Lancellotti P. The role of ischemic mitral regurgitation in the pathogenesis of acute pulmonary edema. *N Engl J Med* 2004; 35:871–873.

60. Pie´rard LA, Lancellotti P. Dyspnea and stress testing. *New Engl J Med* 2006; 354: 871–873.
61. Dickstein K, Cohen-Solal A, Filippatos G, McMurray JJV, Ponitowski P, Poole-Wilson PA et al. ESC Guidelines for diagnosis and treatment of acute and chronic heart failure 2008. *Eur Heart J* 2008; 29:2388–2442.
62. Capomolla S, Febo O, Gnemmi M, Roccardi G, Opasich C, Caporotondi A et al. Betablockade therapy in chronic heart failure: diastolic function and mitral regurgitation improvement by carvedilol. *Am Heart J* 2000; 139:596–608.
63. Vardas PE, Auricchio A, Blanc JJ, Daubert JC, Drexler H, Ector H et al. Guidelines for cardiac pacing and cardiac resynchronization therapy: the task force for cardiac pacing and cardiac resynchronization therapy of the European Society of Cardiology. Developed in collaboration with the European Heart Rhythm Association. *Eur Heart J* 2007; 28:2256–2295.
64. Breithardt OA, Sinha AM, Schwammenthal E, Bidaoui N, Markus KU, Franke A et al. An integrated mechanism for functional mitral regurgitation: leaflet restriction versus coapting force: in vitro studies. *J Am Coll Cardiol* 2003; 41: 765–770.
65. Porciani MC, Macioce R, Demarchi G, Chiostri M, Musili N, Capelli F et al. Effects of cardiac resynchronization therapy on the mechanisms underlying

functional mitral regurgitation in congestive heart failure. *Eur J Echocardiogr* 2006; 7:31–39.

66. Lancellotti P, Meillon P, Sakalihasan N, Waleffe A, Dubois C, Bertholet M et al. Effect of cardiac resynchronization therapy on functional mitral regurgitation in heart failure. *Am J Cardiol* 2004; 94:1462–1465.

67. Madaric J, Vanderheyden M, Van Laethem C, Verhamme K, Feys A, Goethals M et al. Early and late effects of cardiac resynchronization therapy on exercise induced mitral regurgitation:relationship with left ventricular dyssynchrony, remodelling and cardiopulmonary performance. *Eur Heart J* 2007; 28:2134–2141.

68. Kanzaki H, Bazaz R, Schwartzman D, Dohi K, Sade LE, Gorcsan J. A mechanism for immediate reduction in mitral regurgitation after cardiac resynchronization therapy. *J Am Coll Cardiol* 2004; 44:1619–1625.

69. Brandt RR, Reiner C, Arnold R, Sperzel J, Pitschner HF, Hamm W. Contractile response and mitral regurgitation after temporary interruption of long-term cardiac resynchronization therapy. *Eur Heart J* 2006; 27:187–192.

70. Ypenburg C, Lancellotti P, Tops LF, Bleeker GB, Holman ER, Pie´rard LA et al. Acute effects of initiation and withdrawal of cardiac resynchronization therapy on papillary muscle dyssynchrony and mitral regurgitation. *J Am Coll Cardiol* 2007; 50:2071–2077.

71. Aklog L, Filsoufi F, Flores KQ, Chen RH, Cohn LH, Nathan NS et al. Does coronary artery bypass grafting alone correct moderate ischemic mitral regurgitation? *Circulation* 2001; 104(Suppl. I): I-8–I-75.
72. Schroder JN, Williams ML, Hata JA, Muhlbaier LH, Swaminathan M, Matheuw JP et al. Impact of mitral valve regurgitation evaluated by intraoperative transoesophageal echocardiography on long-term outcomes after coronary artery bypass grafting. *Circulation* 2005; 112(Suppl. I): I-293–I-298.
73. Bolling SJ, Pagani FD, Deeb GM, Bach DS. Intermediate-term outcome of mitral reconstruction in cardiomyopathy. *Thorac Cardiovasc Surg* 1998; 115:381–388.
74. Bax JJ, Braun J, Somer ST, Klautz R, Holman ER, Versteegh MIM et al. Restrictive annuloplasty and coronary revascularization in ischemic mitral regurgitation. Results in reverse left ventricular remodeling. *Circulation* 2004; 110: II-103–II-108.
75. Wu AU, Aaronson KD, Bolling SF, Pagani FD, Welch K, Koelling TM. Impact of mitral valve annuloplasty on mortality risk in patients with mitral regurgitation and left ventricular systolic dysfunction. *J Am Coll Cardiol* 2005; 45:381–387.

76. Wong DR, Agnihotri AK, Hung JW, Vlahakes GJ, Akins CW, Hilgenberg AD et al. Long-term survival after surgical revascularization for moderate ischemic mitral regurgitation. *Ann Thorac Surg* 2005; 80:570–577.
77. Diodato MD, Moon MR, Pasque MK, Barner HB, Moazami N, Lawton JS et al. Repair of ischemic mitral regurgitation does not increase mortality or improve long-term survival in patients undergoing coronary artery revascularization: a propensity analysis. *Ann Thorac Surg* 2004; 78:794–799.
78. Mihaljevic T, Lam BK, Rajeswaran J, Takagaki M, Lauer MS, Gillinov AM et al. Impact of mitral valve annuloplasty combined with revascularization in patients with functional ischemic regurgitation. *J Am Coll Cardiol* 2007; 49:2191–2201.
79. Fattouch K, Guccione F, Sampognaro S, Panzarella G, Corrado E, Navarra E et al: Efficacy of adding mitral valve restrictive annuloplasty to coronary artery bypass grafting in patients with moderate ischemic mitral valve regurgitation: a randomized trial. *J Thorac Cardiovasc Surg* 2009; 138: 278–285.
80. Jones HR. Adding mitral valve annuloplasty to surgical revascularization does not benefit patients with functional ischemic mitral regurgitation. *J Am Coll Cardiol* 2007; 22:2202–2203.

81. Vassileva CM, Boley T, Markwell S, Hazelrigg S. Meta-analysis of short-term and long-term survival following repair versus replacement for ischemic mitral regurgitation. *Eur J Cardiothorac Surg*. 2011; 39(3):295-303.
82. Vahanian A, Baumgartner H, Bax J, Butchart E, Dion R, Filippatos G, et al. Guidelines on the management of valvular heart disease. The Task Force on the management of valvular heart disease of the European Society of Cardiology. *Eur Heart J* 2007; 28:230–268.
83. Gisbert A, Soulié`re V, Denault AY, Bouchard D, Couture P, Pellerin M, et al. Dynamic quantitative echocardiographic evaluation of mitral regurgitation in the operating department. *J Am Soc Echocardiog* 2006; 19:140–146.
84. Gelsomino S, Lorusso R, De Cicco G, Capecchi I, Rostagno C, Caciolli S et al. Five-year echocardiographic results of combined undersized mitral ring annuloplasty and coronary artery bypass grafting for chronic ischaemic mitral regurgitation. *Eur Heart J* 2008; 29: 231–240.
85. Hung J, Papakostas L, Tahta SA, Hardy BG, Bollen BA, Duran CM, Levine RA. Mechanism of recurrent ischemic mitral regurgitation after annuloplasty. *Circulation* 2004; 110(Suppl. II): II85-II90.
86. Magne J, Pibarot Ph, Dagenais F, Hachicha Z, Dumesnil JG, Se´ne´chal M. Preoperative posterior leaflet angle accurately predicts outcome after restrictive

mitral valve annuloplasty for ischemic mitral regurgitation. *Circulation* 2007; 115: 787–791.

87. Messas E, Guerrero JL, Handschumacher MD, Conrad C, Chow CM, Sullivan S et al. Chordal cutting: a new therapeutic approach for ischemic mitral regurgitation. *Circulation* 2001; 104:1958–1963.

88. De Bonis M, Lapenna E, La Canna G, Ficarra E, Pagliaro M, Torracca L et al. Mitral valve repair for functional mitral regurgitation in end-stage dilated cardiomyopathy: role of the ‘edge-to-edge’ technique. *Circulation* 2005; 112: I402–I408.

89. Webb JG, Hamek J, Munt BI, Kimblad PO, Chandavimol M, Thompson CR et al. Percutaneous transvenous mitral annuloplasty. Initial human experience with device implantation in the coronary sinus. *Circulation* 2006; 113: 851–855.

90. Sack S, Kahlert P, Bilodeau L, Pie´rard LA, Lancellotti P, Legrand V et al. Percutaneous transvenous mitral annuloplasty: initial human experience with a novel coronary sinus implant device. *Circ Cardiovasc Interv* 2009; 2:277–284.

91. Messas E, Bel A, Morichetti M, Carrion C, Handschumacher MD, Peyrard S et al. Autologous myoblast transplantation for chronic ischemic mitral regurgitation. *J Am Coll Cardiol* 2006; 47:2086–2093.

92. Kunwar B K, Hooda A, Joseph G. Recent trends in reperfusion in ST elevation myocardial infarction in a South Indian tier-3 city. *Indian Heart J.* 2012 Jul-Aug; 64(4):368-73.
93. Jose VJ, Gupta SN. Mortality and morbidity of acute ST segment elevation myocardial infarction in the current era. *Indian Heart J.* 2004; 56:210-214.
94. Darbar D, Gillespie N, Choy AM, Lang CC, Pringle SD, Pringle TH et al. Diagnosing left ventricular dysfunction after myocardial infarction: the Dundee algorithm. *QJM.* 1997; 90(11):677-83.
95. A Marmor, E M Geltman, D R Biello, B E Sobel, B A Siegel and R Roberts. Functional response of the right ventricle to myocardial infarction: dependence of the site of left ventricular infarction. *Circulation.* 1981; 64:1005-1011.
96. Dorosz JL, Lezotte DC, Weitzenkamp DA, Allen LA, Salcedo EE. Performance of 3-dimensional echocardiography in measuring left ventricular volumes and ejection fraction: a systematic review and meta-analysis. *J Am Coll Cardiol.* 2012; 59(20):1799-808.

INFORMATION SHEET

Prevalence and Echocardiographic assessment of Mitral Regurgitation including 3D-EF [Ejection fraction] assessment in Acute ST Elevation Myocardial Infarction

1. This study is for research purpose and to study the prevalence and echocardiographic assessment of Mitral Regurgitation including 3D-EF [Ejection fraction] assessment in Acute ST Elevation Myocardial Infarction
2. Expected duration : 1 year
3. Description of the procedures – ECG and ECHO
4. No foreseeable risks or discomforts to the subject
5. No direct benefit to the subject
6. No alternative procedures available to the subject
7. Complete confidentiality of records identifying the subject will be maintained by Principal Investigator.
8. No compensation available to the subject in the event of a study – related injury
9. No anticipated prorated payment to the subject
- 10.No subjects responsibilities on participation
- 11.Subject's participation is voluntary, that the subject can withdraw from the study at any time and that refusal to participate will not involve any penalty or loss of benefits to which the subject is otherwise entitled
- 12.No apparent foreseeable circumstances under which the subject's participation may be terminated by the investigator without the subject's content.
- 13.No additional costs to the subject that may result from participation in the study
- 14.Subject can withdraw from the research and procedures at any time of study
- 15.There will be no consequences on subjects decision to withdraw from the research
- 16.Subject or subject's representative will be notified in a timely manner if significant new findings develop during the course of the research which may be affect the subject's willingness to continue participation will be provided
- 17.We are not involving pregnant women in our study

18. Approximate number of subjects enrolled in the study – 100 patients

STUDY PROFORMA
DATA ABSTRACTION FORM

DATE OF ENROLMENT:

ENROLMENT NO. :

NAME:

AGE:

HOSPITAL NO.:

ADDRESS:

PHONE:

CLINICAL DIAGNOSIS:

BMI:

HR:

BP:

CLINICAL CHARACTERISTICS	
AGE	
SEX	
DM	
SMOKING	
HTN	
ALCHOLISM	
DYSLIPIDEMIA	
OBESITY	
FAMILY HISTORY OF CAD	
REPERFUSION	
MI	

Age Group

20-39yrs 1

40-59yrs 2

60-79yrs 3

≥80yrs 4

SEX-1[MALE], 2[FEMALE]

RISK FACTOR-1(PRESENT)/2(ABSENT)

MI-1(AWMI)/2(IWMI)

REPERFUSION: 1 (THROMBOLYSIS)/2(PCI)/3(Miscellaneous)

MR SEVERITY:

	DURING HOSPITALISATION	AFTER 1 MONTH
JET AREA 1. PLAX 2. A4C		
VENA CONTRACTA (mm)		
PISA a. PISA radius(cm) b. EROA(cm ²) c. Regurgitant volume(R vol)		
MR SEVERITY		

MR SEVERITY- 0[Absent], 1[Mild], 2[Moderate], 3[Severe]

LEFT VENTRICULAR SYSTOLIC FUNCTION PARAMETERS

	DURING HOSPITALISATION	AFTER 1 MONTH
LVIDd (cm)		
LVIDs (cm)		
2D-EDV		
2D-ESV		
2D-EF		
3D-EDV		
3D-ESV		
3D-EF		

GLOSSARY FOR MASTER CHART

H.NO: Hospital number

AGE (in years)

1= 20-39yrs

2= 40-59yrs

3= 60-79yrs

4= ≥ 80 yrs

SEX: 1=MALE], 2=FEMALE

RISK FACTOR: 1=PRESENT, 2=ABSENT

STEMI: Site of STEMI

1= AWTMI

2= IWTMI

DM: Diabetes Mellitus

HTN: Hypertension

SM: Smoker

DL: Dyslipidemia

FH: Family history of CAD

SITE OF MYOCARDIAL INFARCTION (MI): 1=AWMI, 2=IWTMI

REP: REPERFUSION

1=THROMBOLYSIS), 2=PCI, 3=Miscellaneous

EF: Ejection fraction

LVF: LEFT VENTRICULAR SYSTOLIC (LV) FUNCTION (%)

Normal: 1= $\geq 55\%$

Mild Dysfunction : 2= 45-54%

Moderate Dysfunction: 3= 30-44%

Severe Dysfunction: 4= <30%

BODY MASS INDEX (BMI)

Overweight: 1= 24.9

Normal: 2= 18.4-24.9

Underweight: 3= <18.4

MITRAL REGURGITATION (MR)

0= Absent

1= Mild

2= Moderate

3= Severe

MRJA: MR jet area

LAA: Left atrium area

PLAX: Parasternal long axis view

A4C: Apical 4 chamber view

VC: Vena contracta

RAD: Proximal isovelocity surface area radius

EROA: Effective regurgitant orifice area

PISA: Proximal isovelocity surface area

R Vol: Regurgitant volume

LVIDd: Left ventricular internal diameter in diastole

LVIDs: Left ventricular internal diameter in systole

2D EDV: 2D end-diastolic volume

2D ESV: 2D end-systolic volume

3D EDV: 3D end-diastolic volume

3D ESV: 3D end-systolic volume

2D EF: 2D ejection fraction

3D EF: 3D ejection fraction

MR STATUS

1= Improvement

2= Persistent

3= Deterioration

Master Chart - Baseline characteristics at index hospitalization

	H.No.	Age	Sex	STEMI	DM	HTN	SM	DL	BMI	FH	REP	MRJA(PLAX)	MRJA(A4C)	VC	Rad	ERO	R Vol(ml)	MR	LVIDd	LVIDs	2D EDV	2D ESV	2D EF	LV F	3D EDV	3D ESV	3D EF
1	054103F	47	2	2	1	1	2	1	1	2	2							0	3.8	2.5	43	22	51.2	2	58.6	25.6	56.4
2	238274C	60	1	1	1	1	1	1	2	2	1							0	3.9	2.9	65.9	30.9	53.2	3	63.5	30.4	52.1
3	057118F	55	2	1	2	1	2	1	1	1	3							0	5.4	4.6	109	68	37.6	3	98	56.2	42.6
4	064339F	48	1	1	2	2	1	1	2	2	1	2.4/11.6	2/8.3	2.5	0.2	0.02	2	1	4.9	3.9	113.5	67.4	40.6	3	62.2	29	53.4
5	065890F	72	1	2	2	2	1	1	1	2	1							0	5	4	118.2	70	40.8	3	73.3	42.3	42.3
6	055388F	50	1	2	2	1	1	1	1	2	1							0	4.1	3.1	59	28	52.5	2	58.1	24.6	57.7
7	054675F	55	1	2	2	2	2	1	1	2	1	1.78/10.4	2/14.8	2.6	0.3	0.03	5	1	4.7	3.6	101.5	54.5	46.3	2	97.5	55.2	43.4
8	054634F	43	1	1	2	2	1	1	2	1	2							0	5	3.9	82.5	51.6	37.5	3	76.3	49.9	34.6
9	054488F	49	2	2	2	1	2	1	1	1	2							0	3.7	2.5	56.2	25.3	55	1	74.6	41.3	44.6
10	054408F	41	1	2	2	2	2	1	1	2	2	1.8/15.8	2.1/15.4	2.4	0.3	0.04	6	1	4.9	3.5	70	66	51.5	2	84.5	43.4	48.6
11	054547F	35	1	1	1	2	1	1	2	2	1							0	4.7	3.8	93	51	45.2	2	88.9	47.8	46.2
12	050273F	68	1	1	1	2	2	1	1	2	3							0	4.7	3.6	85	32	62.4	1	80.7	33.2	58.7
13	057169F	37	1	2	2	2	2	1	2	1	3							0	4.2	2.9	76.8	35.3	54	2	84.2	41.4	50.8
14	077453A	48	1	1	1	1	1	1	1	2	2							0	3.6	2.6	54.4	24.6	54.8	2	50.5	22.4	55.6
15	069862F	65	2	2	1	2	2	1	2	2	1	3.7/14.7	4.5/16.5	5.2	0.4	0.07	9	1	4.7	3.6	102.4	54.4	46.8	2	75.2	34	54.7
16	882378D	73	1	2	1	1	2	1	1	2	2	5.8/17.6	6.3/18	4.4	0.6	0.14	24	2	4	3.3	70.4	43.1	38.8	3	69.1	38.9	43.7
17	073346F	50	1	2	2	2	2	1	1	2	1	4.4/13.2	1.6/8.68	2.9	0.4	0.11	15	1	4	2.8	67.9	29.6	56.5	1	59	27.7	53.5
18	077605F	61	1	1	1	2	2	1	1	1	2							0	4.1	2.8	74.2	28.7	61.3	1	63	32.4	48.5
19	077583F	60	2	1	2	1	2	1	2	2	1							0	3.7	2.8	58.1	29.6	49.2	2	57.4	37.9	34
20	225068D	68	1	1	2	1	2	1	1	2	2							0	4.4	3.2	87.7	41.7	52.4	2	69.1	38.9	43.7
21	077403F	49	1	1	2	1	2	1	1	2	2							0	5.1	4	123.8	72.1	41.8	3	74.4	29.9	59.8
22	107333F	49	2	1	2	2	2	1	1	2	2							0	3	2.5	35	22.3	36.2	3	67.9	39.5	41.8
23	107374F	45	1	2	2	1	2	1	1	2	1							0	3.7	2.6	58.1	24.6	57.7	1	39.7	27.8	29
24	107254F	29	1	1	2	2	1	1	2	1	1							0	4.3	3.2	85.4	39.4	53.8	2	70	34.6	50.6
25	107146F	50	2	2	2	2	2	1	1	2	1							0	3.9	2.4	65.9	20.2	69.4	1	82.2	28.3	65.6
26	107169F	55	1	1	2	2	1	1	1	1	1							0	5.2	3.6	129.5	54.4	58	1	62.3	31.2	50
27	102418F	38	1	2	2	2	2	1	1	2	2							0	4.2	3.3	78.6	44.1	43.8	3	84.3	44.8	46.8
28	107046F	32	1	1	2	2	1	1	1	2	1							0	5	3.8	118.2	62	47.6	2	103.6	48	53
29	188849C	56	1	1	2	1	1	1	1	1	2							0	5	4.1	121	76.4	36.9	3	72.9	39.9	45.3
30	107022F	68	1	2	2	1	2	1	1	2	2	1.7/11	1.2/9.5	3.6	0.4	0.07	10	1	4.4	3.5	87.7	50.9	42	3	164	76.6	53.6
31	102288F	42	1	1	1	1	1	1	2	2	2							0	4.1	3.2	76.4	41	46.4	2	56.8	28.7	49.5
32	102275F	59	1	2	2	2	1	1	1	2	2							0	4.8	3.5	107.5	50.9	52.7	2	46.6	27.9	52.2

33	102272F	45	1	1	2	2	1	1	2	2	1							0	4.2	3.1	78.6	37.9	51.7	2	69.7	28.7	58.8
34	102281F	45	1	2	1	1	2	1	2	2	1							0	4.7	3.3	38	17	55.3	1	40.5	28.8	28.8
35	119004F	30	1	1	1	2	1	1	1	2	2							0	4.1	3.2	56.8	34	40	3	50.2	27.6	45
36	112993F	52	2	1	1	1	2	1	1	2	2							0	3.5	2.5	51.6	21.4	58.4	1	64	28	56.2
37	112996F	61	1	2	2	2	2	1	2	2	1							0	4	3.1	67.9	39.4	42	3	75.6	50.8	32.8
38	729726C	36	1	2	2	2	1	1	1	2	2							0	4.5	3.5	94.4	51.4	45.5	2	98.6	60.5	38.6
39	054139F	60	1	1	1	2	2	1	1	2	2							0	4.6	3.8	97.3	62	36.4	3	88	50.68	42.4
40	112859F	52	1	1	1	1	2	1	2	2	2							0	5.7	4.7	160	102.4	36	3	61.9	40.4	34.8
41	118041F	59	1	2	2	2	1	1	1	2	1	3.7/10.9	4/11.7	3.1	0.4	0.06	10	1	6.1	4.3	186.2	85.4	54.2	2	78.9	43.4	45
42	112856F	31	1	2	2	2	1	1	2	2	2	3.6/18	5/17.6	5.2	0.3	0.04	6	1	5.6	4.1	153.7	74.2	51.7	2	103.1	53.4	48.1
43	115998F	43	1	1	2	2	2	1	1	1	2							0	3.4	2.7	47.4	27	43	3	56.8	31.7	44.2
44	110143F	49	1	1	1	1	2	1	1	2	2							0	4	3.1	70	37.9	45.8	2	66.2	26.2	60.5
45	112533F	61	1	2	1	1	2	1	2	2	3	1.7/12.4	1.8/9.5	2.3	0.3	0.03	6	1	4.1	3.2	74.2	41	44.8	2	84	48.6	42.2
46	112611F	66	1	1	2	2	2	1	1	2	1							0	4.3	3.3	54.6	31.7	42	3	58.7	30.8	40.7
47	112508F	39	2	1	2	2	2	1	1	2	3							0	4.4	3.5	89.3	50.9	43	3	39.5	14.2	64
48	102124F	39	1	1	2	2	2	1	2	1	3							0	5.1	4	123.8	70	43.5	3	45.9	31.3	31.8
49	101269F	55	1	2	2	2	1	1	2	2	1							0	3.9	2.7	65.9	27	59	1	51.7	17.8	65.6
50	098767F	51	1	1	2	2	1	1	1	2	2							0	4.2	3.2	78.6	41	47.9	2	72.4	39.7	45.1
51	098764F	50	1	2	2	2	2	1	2	2	2							0	4.5	3.2	90.1	41	54.5	2	72.6	32	55.9
52	098685F	55	1	1	2	2	1	1	2	2	1							0	4.9	4	112.8	70	38	3	78.3	49.2	37.1
53	090246F	45	1	2	2	2	2	1	1	2	3							0	4.2	3.3	78.6	44.1	43.8	3	57.5	30.6	46.8
54	927328D	57	1	1	1	2	2	1	1	1	3							0	4.9	4.1	112.8	74.2	34.2	3	56.7	35.5	37.3
55	662271C	38	1	2	2	2	2	1	2	1	2							0	5.1	4	123.8	70	43.5	3	56.8	31.7	44.2
56	094969F	59	1	2	1	2	2	1	2	2	1	2.6/12.8	3.4/13.6	1.5	0.3	0.04	5	1	4	2.9	70	32.2	54	1	37.1	13.9	62.5
57	098619F	50	2	2	1	1	2	1	2	2	1	3.1/10.6	6.8/12.3	4.6	0.6	0.16	28	2	3.5	2.7	50.9	27	46.9	2	53.7	34.1	36.4
58	098590F	43	1	2	2	2	2	1	1	2	1							0	4.5	3.4	92.4	47.4	48.7	2	75.2	37.3	50.5
59	098521F	52	1	2	1	2	1	1	2	2	2	2.1/13.9	2.6/14.2	2.5	0.4	0.07	4	1	6	4.5	180	90.1	50	2	87.6	56.4	35.6
60	094810F	65	1	1	1	1	1	1	2	2	2	2.3/8.4	1.6/6	2.5	0.2	0.02	2	1	5	4	118.2	70	40.8	3	82.4	57.4	30.3
61	578325C	55	1	2	1	2	1	1	2	2	2	1.7/8	1.4/7.2	3.1	0.4	0.07	8	1	4.9	3.6	112.8	54.4	51.8	2	100.8	46.4	54
62	097655F	60	1	1	1	2	1	1	2	2	2	2.2/12.6	2.5/11.4	2.5	0.3	0.04	4	1	5.4	4.6	141.3	95.7	32.3	3	107.9	71.1	39.1
63	094712F	55	1	2	2	2	2	1	2	2	2							0	4.8	3.3	107.5	44.1	59	1	98.8	43.1	56.4
64	094702F	65	1	2	1	2	1	1	2	2	2	1.9/11.8	2.2/11.3	2.7	0.3	0.04	6	1	4.9	3.8	110.2	62	43.8	3	109.5	58.8	46.3
65	094553F	36	1	2	2	2	1	1	1	1	1							0	4	2.6	67.9	24.6	63.8	1	94.7	44.3	53.2
66	094459F	44	1	2	2	2	1	1	1	2	2	1.8/15.7	1.4/8.6	2.6	0.2	0.02	3	1	4.8	3.2	107.5	41	61.9	1	73.9	37.5	49.3

67	093552F	65	1	2	1	1	1	1	1	2	2	1.3/11	1.2/12	3.5	0.2	0.02	3	1	3.9	2.7	63.9	27.8	56.4	1	56.8	27.1	52.3
68	092687F	60	2	1	2	1	2	1	2	2	1							0	4.5	3.5	92.4	50.9	45	2	64.8	27.1	58.2
69	089713F	65	2	1	2	1	2	1	1	2	1							0	4.3	3.3	80.8	44.1	45.4	2	71.3	42.6	40.2
70	089728F	58	2	2	1	2	1	1	1	2	3							0	4.1	3.1	74.2	37.9	48.9	2	49.7	26.9	45.9
71	089921F	53	2	2	2	2	2	1	1	2	2	2.3/9.1	2.7/8.9	1.9	0.5	0.1	14	1	3.7	2.3	58.1	18.1	68.8	1	50.3	26.7	46.9
72	685616C	51	1	1	2	1	2	1	1	2	1							0	4.9	3.9	112.8	65.9	41.6	3	102.4	57.9	43.4
73	089991F	70	2	2	2	2	2	1	3	2	1	5.3/17.4	5.1/13/	2.6	0.4	0.07	11	1	3.9	2.6	65.9	24.6	62.7	1	78.4	32.8	58.2
74	089887F	65	2	1	2	2	2	1	1	2	1							0	4.1	3.2	68.4	41	40	3	76.4	46.8	38.8
75	088032F	40	1	2	2	2	1	1	2	2	2							0	4.2	3.1	78.6	38.9	50.5	2	75.2	33.1	56
76	089857F	50	2	1	2	1	2	1	1	2	3							0	4.6	3.5	88.2	52.2	40.8	3	97.4	56.4	42.1
77	089599f	78	1	1	2	1	1	2	2	2	2							0	5.5	4.4	146.8	87.7	40.3	3	136.2	82.3	39.6
78	089863F	61	1	2	2	1	1	1	1	2	1							0	4.5	3.4	90.1	49.1	45.4	2	97.3	50	48.6
79	088472F	45	2	1	1	2	2	1	2	2	2							0	2.9	2.3	32.2	18.1	43.7	2	56.7	30.6	46.1
80	089412F	53	1	1	2	1	1	1	2	2	2							0	4.8	3.5	76.2	39.6	48	2	56.9	29.5	48.1
81	089405F	56	1	1	2	2	1	1	2	2	2							0	5.1	4	123.8	70	43.5	3	86.6	46.2	46.7
82	084441F	40	1	2	2	2	1	1	2	2	2							0	5	3.5	118.2	50.9	57	1	82.2	45.1	45.1
83	300574C	59	1	2	1	2	2	1	2	2	2							0	4.4	3	86.1	36.3	57.8	1	88.6	50.1	43.5
84	085060F	43	1	1	2	2	1	1	1	2	1							0	4.1	3.2	74.2	41	44.8	2	114.9	60.1	47.7
85	077488F	67	1	2	1	1	1	1	2	2	2							0	4.9	3.8	112.8	63.9	43.3	3	103.1	74.1	28.1
86	077667F	70	2	1	1	1	2	1	1	2	1	4.3/17.4	5/18.2	3.3	0.4	0.06	9	1	4.2	3.4	78.6	47.4	39.6	3	88.2	44.7	49.3
87	077610F	82	1	1	2	1	2	1	2	2	1	2.4/15.3	2.7/13.8	2.3	0.4	0.07	12	1	5	4.1	121	74.2	38.7	3	61.3	37.8	38.3
88	368074A	77	1	2	2	1	2	1	1	1	2							0	3.6	2.9	54.4	32.2	40.8	3	67.4	38.9	42.3
89	054634F	43	1	1	2	2	1	1	1	2	2							0	4.5	3.6	92.4	54.4	41.1	3	98.3	61.1	37.8
90	080409F	60	1	1	1	2	2	1	1	2	1							0	4.5	3.5	94.9	50.9	46.4	2	53	32.6	38.1
91	328853F	35	1	1	2	2	1	1	2	1	1							0	4.6	3.8	99.8	62	37.9	3	89.7	52.5	41.5
92	331075F	65	1	2	2	2	1	1	2	2	1	1.21/15	1.99/13	2.9	0.2	0.06	8	1	4.5	3.1	92.4	37.9	59	1	90.3	49.2	45.5
93	322267F	47	1	1	2	2	1	1	2	2	1							0	4.1	3.2	74.2	41	44.8	2	47	32.2	31.6
94	495506C	64	1	2	1	2	1	1	1	2	2	1.4/11.9	3.3/17.5	4.9	0.5	0.04	39	2	4.9	3.9	112.8	65.9	41.6	3	110.8	67.6	39
95	322632F	58	1	2	1	1	1	1	1	1	2							0	5.2	4.1	129.5	73.5	43.2	3	132.5	77.8	41.3
96	327344F	80	2	2	1	1	2	1	1	2	2							0	4	3.2	70.1	41.5	40.8	3	64.2	45.5	29.1
97	327352F	65	2	2	2	2	2	1	2	2	2	0.9/14	3.95/16.6	3.1	0.3	0.12	10	1	4.6	3.7	97.8	58.1	40.6	3	67.4	33.2	43
98	322650F	45	1	2	2	2	1	1	2	2	2							0	4.2	3.1	80.1	36.6	54.3	2	62.3	31.8	49
99	322636F	47	1	1	1	1	2	1	1	2	2							0	3.9	2.8	67.9	28.3	58.4	1	72	40.1	44.3
100	433108D	40	1	1	2	2	1	1	1	2	1							0	5	4	121	72.1	40.4	3	76.5	46.8	38.8

101	347512F	63	2	1	2	1	2	1	1	2	3		2.61/14.5(PL	4.9	0.3	0.04	5	1	4.7	3.8	100.7	62	38.5	3	116.1	74	36.3
102	327307F	41	1	2	2	2	1	1	2	2	2							0	5.6	3.9	153.7	65.9	57.1	1	86.8	51.5	40.6
103	288407F	48	1	1	2	2	2	1	2	2	2							0	4.2	3.2	78.4	41.4	47.2	2	84	46	45.2
104	287354F	52	1	2	1	1	2	2	1	2	2							0	3.9	1.2	129.5	65.9	49.1	2	40	20	50
105	291306F	65	1	1	2	2	1	1	2	2	2							0	3.8	2.9	62	32.2	48	2	66.8	35.8	46.5
106	256769D	60	2	1	1	1	2	1	1	2	2							0	4	3.2	70	41	41.5	3	100	57.5	42.5
107	286849F	46	1	1	2	1	1	1	2	2	2							0	5	4.2	118.5	78.6	33.7	3	86	56.3	34.5
108	299314F	56	1	2	2	1	2	1	2	2	2							0	4.5	3.2	92.4	40.3	56.4	1	88.7	37.3	58
109	299336F	82	1	2	2	2	2	1	1	2	2							0	4.9	3.6	112.3	55.5	50.5	2	114.6	59.13	48.4
110	309758F	45	1	1	2	1	2	1	2	2	2							0	3.8	3	62	35	43.5	2	56	33	41.1
111	761606D	62	1	2	1	1	1	1	2	2	2							0	4.3	3.2	85.4	41	52	2	73	35.6	51.3
112	981786A	47	1	2	2	2	2	1	2	2	2							0	4.8	3.7	107.5	58.1	45.9	2	118	65	44.9
113	316403F	48	1	2	2	2	1	1	3	2	2							0	5.2	4.1	128.7	76.3	40.7	3	75	44	41.3
114	316458F	50	1	1	2	1	1	1	1	2	2							0	3.8	2.8	62.4	29.6	52.6	2	44	18	59.1
115	040901F	45	1	1	2	2	1	1	1	2	3							0	4.7	3.1	103.9	39.1	62.3	1	85.9	30.92	64
116	077397F	56	1	2	2	2	1	1	1	2	1							0	4.3	3.1	85	45	47.1	2	76	40.6	46.6
117	089817F	70	1	2	2	2	1	1	2	2	2							0	4.7	3.7	39	23	41	3	54.6	34	37.8
118	354481F	50	1	1	2	1	1	1	1	2	1							0	5.3	3.7	88	36	59.1	1	60.7	25.2	58.2
119	354493F	55	1	1	2	2	2	1	1	2	1							0	3.7	3	74	43	41.9	3	70.1	41.9	40.3

Follow-up data after 1 month

	H.No.	MRJA(PLAX)	MRJA(A4C)	VC	Rad	ERO	R Vol(ml)	MR	LVIDd	LVIDs	2D EDV	2D ESV	2D	LV F	3D EDV	3D ESV	3D EF
1	054103F							0	3.6	2.6	54.4	24.6	54.8	2	50.5	22.4	55.6
2	238274C							0	4.5	3.2	90.1	41	54.5	2	66.2	26.2	60.5
3	064339F							0	4.9	3.5	70	66	51.5	2	76.4	33.1	56.7
4	065890F							0	5.3	4.2	180	113	37.2	3	104.3	68.6	34.2
5	055388F							0	3.9	2.7	63.9	27.8	56.4	1	80.5	27.5	65.9
6	054675F							0	5	3.8	118.2	62	47.6	2	46.6	27.9	52.2
7	054634F							0	3.8	2.9	66	32	51.6	2	81	38.8	52.1
8	054408F							0	3.7	2.8	58.1	29.6	49.2	2	88.9	47.8	46.2
9	077453A							0	3.7	2.6	58.1	24.6	57.7	1	58.6	25.6	56.4
10	069862F	1.8/11	1.6/15	3.1	0.4	0.06	10	1	4.7	3.5	80	38	52.5	2	61.1	20.2	67
11	882378D							0	3.7	2.8	58.1	29.6	49.2	3	87.4	45.7	48
12	073346F							0	3.5	2.4	68	26	61.8	1	59	26.3	55.5
13	077605F							0	4.3	2.9	65	24	63.1	1	71.2	35.4	50.3
14	225068D							0	3.7	2.6	60.4	26.6	56	1	78.7	32.8	58.4
15	107254F	1.8/11	1.9/10.7(A4C)	2.6	0.4	0.07	13	1	4.5	3.1	81	43	59	1	86.3	42.5	50.8
16	107146F	4.3/15.4	5.45/16(A4C)	3.5	0.3	0.04	7	1	4.1	2.9	120	43	56.6	1	113.6	63	44
17	107169F							0	5.2	3.6	129.5	54.4	58	1	64	28	56.2
18	102418F							0	4.7	3.8	75	42	44	3	75.7	38.6	49.1
19	107046F							0	4.8	3.5	107.5	50.9	52.7	2	74.4	29.9	59.8
20	188849C							0	6.4	5.1	124	81	34.7	3	91.4	52	43.2
21	107022F	2.4/8.4	2.8/7.44(A4C)	3.4	0.3	0.04	5	1	5.6	3.4	79	24.5	69.1	1	56.2	28.9	48.6
22	102288F							0	5	3.8	62	28	54.8	2	62	29.2	52.9
23	102275F							0	4.7	3.3	38	17	55.3	2	72.6	32	55.9
24	102272F							0	4.9	3.3	63	26	58.7	1	102.1	45.8	55.2
25	102281F							0	5.4	3.7	76	39	48.7	2	63.8	39	38.8
26	119004F							0	4.5	3.6	111	66	40.5	3	88.9	52	41.5
27	112996F	1.4/13.8	1.2/14.2(A4C)	2.3	0.3	0.04	5	1	4.9	3.8	97	55.3	43	3	63.6	36.9	42
28	118041F	2.5/11.6	3.8/12.8(A4C)	2.1	0.2	0.01	2	1	5.6	4.2	158	91.64	42	3	113.4	58.97	40.8
29	112856F	6.7/22	7.7/22.5(A4C)	5.1	0.5	0.1	10	1	5.6	4.8	163	110.84	32	3	146.6	106.2	27.6
30	115998F							0	5.2	4.2	105	63.74	39.3	3	93.1	55.3	40.6
31	110143F							0	4.8	3.2	146	65.7	55	1	58.7	27	54
32	112533F							0	4.5	3.2	90.1	41	54.5	2	78.4	32.8	58.2

33	112611F							0	4.7	3.5	74	26	64.9	1	148.9	76.3	48.7
34	098767F							0	4.2	2.6	70	28	60	1	75.4	38	49.6
35	098764F							0	4.6	3.4	131	61	53.4	2	55.9	32.4	42.1
36	098685F							0	4.5	3.4	68	30	55.9	1	105.6	43.6	58.7
37	090246F							0	4.2	3.3	78.6	44.1	43.8	3	57.5	30.6	46.8
38	927328D							0	4.9	4.1	98.6	64.9	34.2	3	66.7	43.4	35
39	662271C							0	4.4	2.6	79	45	43	3	78.6	42.9	45.4
40	094969F	2.3/8.6	2.2/11.4	2.5	0.4	0.07	4	1	4.3	3.3	80.8	44.1	45.4	2	71.3	42.6	40.2
41	098590F							0	3.5	2.7	50.9	27	46.9	2	53.7	29.6	44.8
42	098521F							0	5.2	4.1	120	63	47.5	2	90	58	35.6
43	578325C	3.9/15.2	4.53/16.2	4.2	0.5	0.11	26	2	4.9	3.5	93	52	44	3	84.7	52.4	38.1
44	094712F							0	5.5	3.4	120	68	43.3	3	58.5	31.8	45.6
45	094702F							0	5.3	4	98	48	51	2	131.6	100	24
46	094553F							0	4.2	2.8	88	38	56.8	1	80.9	32.5	59.8
47	094459F							0	4.4	3	86.1	36.3	57.8	1	69	28.4	58.8
48	093552F							0	4.8	3.2	107.5	41	61.9	1	74.2	28.7	61.3
49	089728F							0	4.7	3.4	89	22	75.3	1	71	38.7	45.4
50	089921F							0	4.1	2.5	90	34	62.2	1	56.7	20.4	64
51	685616C							0	4.1	2.7	59	31	47.5	2	67.4	35	48
52	089887F							0	4.1	3.2	68.4	40	40	3	80	48.9	38.8
53	088032F							0	4.7	3.4	81	36	55.6	1	69.1	34.7	49.8
54	089857F							0	5.3	4	86	38	55.8	1	82.7	38	54.1
55	089599f							0	5.2	3.5	129.5	52	59.8	1	66.6	38.2	42.6
56	088472F							0	3.5	2.5	89	39	56.2	1	57.9	23.7	59.1
57	089412F							0	5.3	4	66	23	65.2	1	69	28.4	58.8
58	089405F							0	4.5	2.9	116	35.1	46	2	65.4	35.2	46.2
59	300574C							0	4.4	3.2	65	36	44.6	3	62.8	35.7	43.1
60	077488F							0	5.5	4.5	93	58.04	37.6	3	95.5	63.3	33.7
61	077667F	3.09/9.68	4.23/15.7	2.4	0.3	0.03	6	1	5.5	4.4	146.8	87.7	40.3	3	61.3	37.8	38.3
62	368074A							0	4.1	2.7	67	31	53.7	2	60.9	31.3	48.6
63	054634F							0	4.5	3.5	94.9	50.9	46.4	2	53	32.6	38.1
64	080409F							0	4.5	3.8	89.8	54.4	39.4	3	89.7	52.5	41.5
65	328853F							0	4.5	3.1	88.6	36.3	59	1	87.8	47.8	45.5
66	331075F	2.35/12.4	2.35/9.22	3.2	0.4	0.06	9	1	4.2	3.3	132	72	45.5	2	79.4	35.7	55.1

67	322267F							0	4.8	3.4	107.5	47.4	55.9	1	60.9	25.5	58.2
68	322632F							0	4.2	3.2	78.4	41.4	47.2	2	84	46	45.2
69	327352F	1.8/14	2.5/15.8	3.1	0.3	0.12	10	1	4.1	3.2	74.2	41	44.8	3	86.8	51.5	40.6
70	322650F							0	4.6	3.3	97.3	44.1	54.7	2	73	43.7	40.2
71	433108D							0	5.2	4	129.5	70	45.9	2	68.2	44.8	34.3
72	347512F							0	4.8	3.6	107.5	56.3	47.7	2	73.7	42.1	42.9
73	288407F							0	4.5	3.4	92.4	47.4	48.7	2	63	33	47.6
74	287354F							0	5.7	4.5	163.1	94.3	42.2	3	48	27	43.8
75	291306F							0	4	2.9	71	32	55	1	82.6	40	54
76	256769D	2.3/17.8	2.8/18.2	2.4	0.3	0.04	6	1	4.6	3.4	97.8	47.4	51.5	2	87.8	48.9	44.3
77	286849F							0	6.2	4.9	194	115	41	3	188	108.8	42.1
78	299314F	4.7/14.8	5.6/18.3	5.4	0.6	0.14	28	2	4.4	3.1	87.8	37.9	56.8	1	92.2	41.1	55.4
79	299336F							0	4.1	2.9	73	33	55	1	78.4	37.3	52.4
80	309758F							0	5.6	4.2	155.8	80.1	48.6	2	76	39	48.7
81	761606D							0	4	2.8	70	29.6	57.8	1	89	44	50.6
82	981786A							0	4.8	3.6	109.6	52.8	51.8	2	82	34	58.5
83	316403F							0	5.6	4.4	151	87	42.4	3	76	44	42.1
84	316458F							0	4	2.9	70	32.2	54	1	87	38	56.3
85	040901F							0	4.9	3.3	112.8	44.1	60.9	1	85	41	51.8
86	077397F							0	4.1	3.1	56	21	62.5	1	93.8	32.2	56.4
87	89817F							0	4.3	3.2	83	39.4	52.5	2	47.9	16	66.5
88	354481F							0	4.3	3.2	81	38	53.1	2	81.6	46.8	42.6
89	354493F							0	5	3.4	112	54	51.8	2	79	35.55	55



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http://catalogue.iugm.qc.ca/GEIDFile/24807.PDF?Archive=103204692148&File=24807_PDF**paper text:****INTRODUCTION****1MR is a frequent complication of coronary artery disease. It**

was often under-rated because it is clinically silent, but with the use of echocardiography this complication

4is observed between 15%-20% after a myocardial infarction.

1

1When compared with patients without MR, the patients with acute myocardial infarction and mitral regurgitation are older, more often in female and frequently have a history of previous

ischaemic heart disease.² Its presence and degree have major prognostic implications and underscore the importance of its detection and quantification. Ischaemic mitral regurgitation can independently predict cardiovascular death with a relative risk of 2.3 In a community trial, Bursi et al found that mitral

regurgitation predicted heart failure as well as mortality among 1 month survivors independent of age, gender, ejection fraction, and Killip Class.⁴ Even mild MR was shown to increase the mortality in SAVE trial (Survival and Ventricular Enlargement) ³ hence, detection and quantification of MR is crucial. MR can be estimated by different techniques on echocardiography. Color flow imaging allows an easy visualization of MR, but was found to overestimate the MR.⁵ McCully et al demonstrated that the same jet area corresponds to smaller regurgitant volumes in functional MR as compared to organic MR.⁵ Doppler echocardiography allows accurate assessment of regurgitant volume and effective regurgitant orifice and thus provide the tools to reliably evaluate the prognosis and mechanism. Doppler methods are simple, fast, reproducible and proven to be more reliable but sparsely applied in routine clinical evaluation. Current guidelines advocate quantification of MR by the measurement of

7vena contracta and proximal isovelocity surface area (PISA),

the most recommended quantitative approach whenever feasible. The semi-quantitative evaluation should be abandoned.⁶ Echocardiography is the most common method to assess left ventricular systolic function. It can efficiently predict the outcome and help in determining the treatment modalities like CRT-D implantation etc. Routine 2D EF measurement has several limitations in AMI patients due to problems of foreshortening and geometric assumptions. 3D echo LVEF is much more accurate especially in presence of regional wall-motion abnormalities as it does not have geometric assumptions and is found to have comparable with present day “gold standard” for systolic function, cardiac MRI.⁷ It can automatically calculate ejection fraction and left ventricular mass using the automated softwares, and is therefore more reproducible. It is several folds accurate as compared to conventional echo. As there is no data for prevalence of Mitral Regurgitation in Indian population, this study was undertaken. We evaluated the prevalence and degree of Mitral Regurgitation in

4the acute phase and after 1

month of STEMI. AIMS AND OBJECTIVES PRIMARY AIM a. To study the following parameters

4in Acute ST Elevation Myocardial Infarction[STEMI] patients

- i. Prevalence of mitral regurgitation[MR] by echocardiography in acute MI and during 1 month follow up
 - ii. 3D-EF(ejection fraction) assessment
- SECONDARY AIM a. Correlation of 2D-EF, 3D-EF and MR in STEMI patients
- REVIEW OF THE LITERATURE

13INTRODUCTION Mitral regurgitation (MR) is defined as systolic retrograde flow from left ventricle (LV) to left atrium

(LA) because of pressure gradient between the two chambers. ⁸ The

1term ischaemic MR does not necessarily imply the presence of true myocardial ischemia, it is in fact an abridgment, characterizing a clinical situation corresponding to chronic coronary artery disease with frequently a prior history of one or more myocardial infarctions leading to progressive global or regional pathological LV remodelling, usually in the absence of reversible ischaemia. ⁹ Ischemic MR is a type of

secondary/functional MR due to coronary artery

1disease. Secondary MR is defined as functional MR, due to LV remodelling by idiopathic cardiomyopathy or coronary artery disease.

8, 10

1It is important to distinguish between primary MR due to organic disease of one or more components of the mitral valve apparatus and secondary MR which is not a valve disease, but represents the valvular consequences of a LV disease.

1There are however limitations in both terms: functional and ischemic. Indeed, recent studies have demonstrated evidence of structural changes in the mitral leaflets in response to tethering on them by LV pathological remodelling. The leaflet adaptation includes enlargement and increased stiffness.

¹¹ PREVALENCE MR is a frequent accompaniment of ischaemic heart disease. Clinical presentation is variable from silent to severe MR presenting with hemodynamic instability. It may be an incidental finding on echocardiography or catheterization. ³ Its importance was often underscored because of low murmur intensity but with the use of echocardiography, MR is observed between 15%- 20% of patients with acute myocardial infarction. ¹ Different investigators have found variable incidence of MR in acute MI with the help of different imaging modalities. Lehmann et al found 13 % incidence of mitral regurgitation early in the course of acute myocardial infarction with the help of contrast left ventriculography. ² Tchong et al reported incidence of post-infarction mitral regurgitation was to be 17.9% of patients within hours of infarction. ¹² Indeed, when it is sought by doppler, MR has been reported to occur in up to 39% of patients with MI. ^{13,14} With the recent advances in non-invasive doppler echocardiography, it is possible to accurately assess the regurgitant volume and effective regurgitant orifice. Old age, diabetes, past history of MI, severe CAD are more frequently associated with STEMI with MR than patients with STEMI without MR. ²,

12, 15 Its presence and degree have major prognostic implications and mandates its detection and quantification. PATHOPHYSIOLOGY Normal mitral valve function depends on perfect delicate interplay

2between the mitral leaflets, the subvalvular- apparatus (which includes: **chordae tendineae and papillary muscle**), **the mitral annulus, and the left ventricle.**

Pathophysiology of ischemic MR is perplexing. Myocardial damage and LV dysfunction usually precedes MR. Ischemic MR is characterized by normal leaflets and subvalvular apparatus and occurs due to restricted motion of the leaflets. According to the Carpentier's classification, ischaemic mitral regurgitation is classified as type 3. 16 Given below is the classification based on motion of leaflet

6in relation to the mitral annular plane (figure 1) 1: **normal leaflet motion.**

Perforation of the leaflet due to traumatic injury or endocarditis, or annular dilatation, which may cause left ventricular disease, is the cause of MR in type1. 2:

9excessive leaflet mobility accompanied by displacement of the free edge of one or both leaflets beyond the mitral annular plane

into the left atrium.degenerative cardiac diseases may cause leaflet prolapse. 3: leaflet restriction. It is further subclassified into 2 varieties:

63a, where the restriction occurs throughout the cardiac cycle, i.e. both in systole and diastole due to shortening **of** the chordae **and/**

2or leaflet thickening such as in rheumatic disease,

63b, where the leaflet restriction is seen in systole only (usually the result of regional wall motion abnormalities seen in ischaemic mitral regurgitation).

16 Figure 1: Carpentier's functional classification of Mitral Regurgitation17 Mitral Leaflet closure is mainly an intricate interplay between the forces of tethering and ventricular forces (figure 2, 3). Increase in tethering forces will not allow adequate

1closure of the mitral leaflets. 8, 18 The

most frequent pattern seen is posterior infarction, more commonly trans mural,

1leading to local left ventricular **pathological remodelling and** thereby **contributing to** posterior, **apical and lateral displacement of the posterior papillary muscle**. As the **papillary muscle** extends **non-extensible** chordate to **both the leaflets, its displacement results in a more apical tethering of the leaflets and their coaptation point, and a characteristic deformity of the anterior leaflet** which is **described as the 'seagull sign'**. 8, 19 **The tethering process produces the shape like that of a tent between the annular plane and the displaced leaflets. The tenting volume closely estimates the regurgitant orifice area.8,**

20 Tenting area is asymmetric in case of posterior infarction and regional remodelling,

1predominates on the posterior leaflet close to the medial commissure. It is accompanied by decreased **mobility of the posterior leaflet. In** another subgroup of **patients**

1with previous anterior infarction **or both anterior and posterior infarctions,**

1LV dilatation is more global, LV is more spherical, both papillary muscles are displaced, the tenting area is symmetric, the regurgitant jet is central and the contribution of annular dilatation and flattening is more important.

Second important determinant is decrease in ventricular closing forces, includes

1altered systolic annular contraction ,LV dysfunction ,**reduced synchronicity between the two papillary muscles and global LV dyssynchrony, especially in basal segments.**

8Factors aggravating the mitral regurgitation are

dilatation of the mitral annulus and the decrease in systolic annular contraction, but isolated annular dilatation does not create functional mitral regurgitation.

18 Figure 2: Left figure: Normal coaptation is seen. Right figure: tethering of leaflet due to papillary muscle displacement and annular dilatation

AO, aorta; Inf PM, inferior papillary muscle; LA, left atrium; LV, left ventricle; MR, mitral regurgitation. (Reproduced from

Levine RA, Hung J, Otsuji Y, et al. Mechanistic insights into functional mitral regurgitation. Curr Cardiol Rep 2002; 4:125–9)

Occasionally, leaflet prolapse can occur as a result of fibrotic elongation of papillary muscle which may be followed by an event such as myocardial infarction and this can result in MR. (Carpenters' Type 2). The consequences of MR depend on the following underlying factors a. Severity of regurgitation b. LA compliance c. LV-LA gradient (the driving force) d. Duration of the lesion Acute MR can occur secondary to two rare causes:

1rupture of a papillary muscles resulting from **acute myocardial infarction and**

transient active ischemia leading to true ischemic MR. The

1rupture of a papillary muscle, more often in

the location of

1head of the posteromedial papillary **muscle, is a catastrophic complication of**

actual MI

1with a high mortality if emergency **surgery is not**

done.^{8, 21} In majority of patients with chronic ischemic MR complicating left ventricular dysfunction and heart failure, LA is enlarged and has a greater compliance with low driving force. The volume overload

resulting from MR contributes to a vicious cycle : the more remodelling of left ventricle, the more severe MR begets MR, so greater the severity of MR, larger the volume overload on LV which finally leads to LV remodelling. LV becomes more and more dilated and spherical in accordance to laplace law, which further propentiates the MR. Despite a reduction in LV impedance, LV wall stress increases ,finally translating into LV dysfunction.²² Chronic ischaemic MR finally leads to development of

1 pulmonary arterial hypertension. Another important feature of ischaemic MR is its

dynamicity. ²³ The

1 degree of MR is best quantified **by the effective** regurgitant **orifice (ERO) area.**
²⁴ **The regurgitation area**

keeps on changing throughout the systole due to dynamic changes in transmitral pressure, though it is of lesser importance in midsystole when compared to other phases of systole.^{25,26} ERO is load dependent, therefore affected by daily activities. Another evidence of the dynamicity of ischaemic MR ↑ tethering forces ↓ Ventricular forces (left ventricle dysfunction) Ischemic MR LA Enlargement ↑ Left Ventricular wall stress AF, PAH left ventricle Remodelling (Annular dilation, papillary muscle displacement) Figure 3: Pathophysiology

1 of Ischaemic MR is a reduction of regurgitant volume related to a reverse LV remodelling obtained by appropriate medical treatment.

²⁷ Dynamic nature of MR can be very well

1 appreciated during an exercise doppler echocardiogram. ²⁸ The degree of MR seen at rest is not related to exercise-induced changes in ERO area or regurgitant volume.

²⁹ Exercise-induced changes are quite variable in different individuals. In some individuals

1 with moderate or severe MR at rest, a decrease in ERO area can be observed with exercise and usually results from contractile reserve of the LV, in particular of the postero-basal segment and/or a reduction in intra LV

dyssynchrony.³⁰ Around

130% of these patients develop an increase in

the severity of regurgitation

1and in systolic pulmonary artery pressure during exercise. The degree of exercise-induced increase or decrease in MR relates to changes in valve deformation, LV remodelling and

papillary muscles synchronicity. DIAGNOSIS AND QUANTIFICATION Clinical examination in ischaemic MR may reveal a regurgitant murmur of low grade or may even be silent or inaudible .It is an insensitive method for ischaemic MR diagnosis due to subtle or near normal auscultatory findings in many patients. This can be explained from the fact that cardiac output is low, LV contractility is compromised and atrial pressure is high thereby they cause lower regurgitant volume, consequently low grade murmur in ischemic MR compared to organic MR. There is no correlation between intensity of murmur and severity of ischaemic MR. The

1diagnosis of ischaemic MR is usually made by using imaging

modalities like doppler echocardiography. Doppler echocardiography is a useful tool in diagnosis and is superior to other techniques like contrast ventriculography. Importance of doppler echocardiography in routine clinical practice is undisputed. Clinically subtle findings like low intensity murmurs

3should always lead to a careful echocardiographic examination. Quantification of MR **is**

also crucial. Echocardiography plays a key role not only in diagnosis of regurgitant lesion but also in the assessment of the mechanism and the severity of MR. It also has a role in determining treatment options as it helps in determining the feasibility of valve repair versus replacement. Assessment of Mitral Regurgitation by echo can be (a) qualitative (b) quantitative. QUALITATIVE METHODS

2Colour flow imaging Colour flow imaging is the most widely used method to assess MR severity

echocardiographically .32 This measurement is poorly reproducible and influenced by various factors. Though it allows an easy visualization of the regurgitation and frequency of MR, it has major limitations in assessing the severity of MR. 5, 33 It tends to overestimate the severity of regurgitation. It is generally assumed that with increasing MR, the

5size and extent of the regurgitant jet in LA

is increased.

2Larger colour jets extending deep into LA represent greater severity of MR than small thin jets that appear just beyond the mitral leaflets. However, the jet size

may be influenced by other factors such as technical and hemodynamic influences and therefore caution must be exercised in interpretation of jet severity based on jet size. For the same severity of MR ,

5patients with increased LA pressure, enlarged LA or wall hugging eccentric

jet may show

2smaller jets area when compared with normal LA pressure, size or with central jets.

6

2In acute MR, even centrally directed jets may be misleadingly small.

Nevertheless,

2a large eccentric jet adhering, swirling and reaching the posterior wall of the LA

favours significant MR and smaller thin jets appearing

5just beyond the mitral leaflets usually

is an indicator of mild MR. Continuous Wave Doppler of MR jet

5Continuous wave doppler of mitral regurgitation jet

is another qualitative parameter which is used to measure the severity of mitral regurgitation.

2The signal intensity (jet density) of the CW envelope of the mitral regurgitant jet can be used as a qualitative indicator to mitral regurgitation severity. A dense triangular mitral regurgitation signal with a full envelope indicates severe mitral regurgitation than a faint signal.

2Truncated (notch) envelope with a triangular contour and an early peak velocity (blunt) indicates elevated LA pressure or a prominent regurgitant pressure wave in the LA due to severe MR. In case of eccentric mitral regurgitation; it may be difficult to record the full CW envelope of the jet,

while the density may be used as indicator for assessing the severity. SEMIQUANTITATIVE MEASURES

1The semi-quantitative evaluation of regurgitant jet area should not be

used.⁶ It is recommended that these measures should only be used for diagnosing mitral regurgitation, and not to quantify the severity of MR.

2A more quantitative approach is recommended when more than a small central MR jet is seen. Vena contracta width (VCW) The vena contracta is the narrowest portion of the MR jet downstream from the orifice; it reflects the effective orifice area.

34-36 Whenever feasible measure the dimensions of vena contracta, which can help in quantification of MR. Using a careful probe angulation and

2adapted Nyquist limit (colour Doppler scale) (40–70 cm/s), the

2vena contracta is typically imaged in a view perpendicular to the commissural line (e.g. the parasternal long-axis or the apical four chamber view)

to

2identify the neck or the narrowest portion of the jet. This narrowest doppler colour sector scan can be coupled with the zoom mode to improve resolution and

for more accurate measurement (Figure 4). It is recommended that if possible averaged measurements over at least three beats should be taken and measurements should be taken from two orthogonal planes. A vena contracta, < 3 mm is considered as an indicator of

2mild MR whereas a width ≥ 7 mm defines severe Mitral Regurgitation, values

in between 3 to 7 mm are ambiguous and need further confirmation by a more quantitative method.⁶ The

2concept of vena contracta is based on the assumption that the regurgitation orifice is almost circular,

this assumption holds true in cases of organic MR but in functional MR the results may not be accurate as the orifice may be non circular and

5elongated along the mitral coaptation line.

37, 38 Thus, it could look

2narrow in four-chamber view and broad in two-chamber view. Conventional 2D colour Doppler imaging does not provide appropriate orientation of 2D scan planes to obtain an accurate cross-sectional view of the vena contracta. The vena contracta can be classically well identified in both central and eccentric jets. In case of multiple MR jets, the respective widths of the vena contracta are not additive. Such characteristics may be better appreciated and measured on 3D echocardiography. In cases of functional MR, a mean vena contracta width (four- and two chamber views) has been shown to be better correlated with calculation done using the 3D vena contracta.

41 3D echo

5assessment of the vena contracta is

not used routinely and currently it is used for research purposes.

2A mean value >8 mm on 2D echo has been reported to define severe MR for all

5aetiologies of MR including functional MR,

though it needs confirmation in further studies.³⁹ Figure 4. Measurement of Vena contracta width in Mitral Regurgitation. Pulmonary venous flow Doppler It is an additional parameter for evaluation of MR severity. Normally, if there is no diastolic dysfunction in venous flow Doppler we get

2a positive systolic wave (S) which is followed by a smaller diastolic wave (D).

As the

2severity of MR increases, there is a blunting of the S wave velocity.

Systolic blunting or systolic flow reversal will be seen in moderate and severe MR respectively.

QUANTITATIVE METHODS

5Doppler volumetric method Doppler volumetric method can

calculate regurgitant volume by finding the difference between measured mitral and aortic stroke volume.⁴³ It can be inferred using 2D echocardiograph by calculation of LVEDV and LVESV which are calculated using biplane method of disks. ⁴² Same jet areas correspond to smaller RVol in ischemic MR than in organic mitral regurgitation. However, this method is cumbersome and time consuming. It is not recommended as first line investigation for quantification of regurgitant volume. Flow Convergence Method Flow-convergence method is the most frequently quantitative method for estimation of MR in current practice.⁴⁴ It is based on law of conservation of flow; its basis lies in the modified form of continuity equation. It enables the measurement of ERO area and regurgitant volume with precision.⁴⁵ As the flow convergence is proximal to the regurgitant orifice it forms the basis of analysis.⁴⁶

2The apical four-chamber view is classically recommended for enabling good visualization of the proximal isovelocity surface area (figure 5). However,

in

5 **anterior mitral valve prolapse the** calculation of

PISA should be done in the parasternal long- or short-axis view. By lowering the image

2 **depth and reducing the Nyquist limit to 15–40 cm/s,**

flow velocity at a hemispheric surface proximal to regurgitant orifice can be determined.⁶ The

2 **radius of the PISA is measured at mid-systole using the first aliasing. Regurgitant volume (R Vol) and effective regurgitant orifice area (EROA) are obtained using the standard formula.**

Regurgitant flow = 2π (radius of the flow convergence)² x aliasing velocity ERO is the ratio of regurgitant flow / peak mitral regurgitant velocity Regurgitant volume (RVol) is calculated as product of ERO and MR TVI.

2 **Qualitatively, the presence of flow convergence at a Nyquist limit of 50–60 cm/s is an indicator to the presence of significant MR. 6 Grading of organic MR**

6 Mild Moderate Severe EROA (mm²) <20 20–29; 30–39

2 **≥40 R Vol (mL) <30 30–44; 45–59**

≥60 Moderate regurgitation group can be further classified

2 **into ‘mild-to-moderate’ (EROA of 20–29 mm², R Vol of 30–44 mL) and ‘moderate-to-severe’ (EROA of 30–39 mm² or R Vol of 45–59**

2 **mL). In ischaemic MR, the thresholds of severity, which are of prognostic value, are 20 mm² and 30 mL, respectively. 47 EROA is the most robust parameter as it represents a marker of lesion severity. A large EROA can lead to large regurgitant kinetic energy (large R Vol) as well as to potential energy, with low R Vol but high LA pressure.**

Ischemic MR Low Risk EROA<20mm² RVol<30ml High Risk EROA>20mm² RVol>30ml Figure 5: Ischaemic MR Grading (6, 48) This method is simple, fast, and reproducible and has been validated by multiple investigators.^{46; 49} It precisely calculates the RVol, which indicates the volume overload induced by MR, and the effective regurgitant orifice (ERO), which delineates the severity of anatomic lesion. PISA radius Figure 5. Determination of radius of proximal isovelocity surface area (PISA) in ischaemic Mitral Regurgitation.

1 There are several limitations of the proximal isovelocity surface area (PISA) approach. ^{8,46} First, the PISA radius changes during systole is larger in early and late systole, and smaller in midsystole when the LV pressure is maximal. ⁵⁰ Ideally, the PISA radius should not be measured at only one time point, but averaged through systole. Second, for an accurate measurement, the flow convergence should be hemispheric.

In cases of functional MR, the

1 flow convergence—a three-dimensional structure—is frequently hemielliptic (Fig 6), implying an underestimated calculation of ERO and regurgitant volume,

particularly

2 when the ratio of long-axis length to short-axis length of the 3D regurgitant orifice is >1.5.

^{38, 51-52} Third, multiple jets can

1 be present; the addition of several flow-convergence regions has not been validated.

Fourth, it is more accurate for central jet. It

2 may not hold for eccentric jets, several jets, or complex or elliptical regurgitant orifices. Practically, the geometry of the PISA varies based on the shape of the orifice and mitral valve leaflets surrounding the orifice.

Figure 6: 3Dimensional

5shape of the convergence flow in (a) functional

MR (b) organic MR (Reproduced from EHJ guidelines

1for the assessment of the valvular regurgitation

2010)

1Thus, practically, the most reliable calculation of regurgitant volume and ERO area is the averaging of the quantitative doppler and the PISA methods

.However, this approach is time consuming.8 Invasive assessment Mitral regurgitation could be assessed by invasive ventriculography. Some schools of thought have considered Left ventriculography

3as the reference methodology for assessment of mitral regurgitation. However this

method of grading of MR is also subject to limitations and cannot thereby be considered gold standard because of influence of loading conditions affecting the MR severity. Comparatively, Quantitative Doppler echocardiography is non invasive and also provides more objective data for grading and prognostic information about regurgitation than ventriculogram.53 PROGNOSIS The presence of ischaemic MR

4is an important indicator for long term

morbidity as well as mortality. It

4is an independent risk factor for the same. The presence of

ischaemic MR may result from acute infarction resulting in regional left ventricular dilation and consequent loss of contractile mechanism or in some instances it may be a previously existing lesion which went undiagnosed. Several studies have clearly shown that ischaemic mitral regurgitation can predict cardiovascular mortality independently.2-4,8, 12, 54 Relative risk was found to be quite variable ranging from 1.48 - 7.5.8

1Worse long term prognosis was seen **in patients** presenting **with non ST elevation**

myocardial infarction .55 A community based

1study has also confirmed the prognostic importance of ischaemic **MR**

among one month survivors of MI:

1its presence is associated with a three-fold increase in the risk of heart failure and a 1.6-fold increased risk of death at 5-year follow-up, independent of LV ejection fraction, Killip class, age and gender.

4,8 Barzilai et al found that AMI patients with a murmur suggestive of MR had a 12-month mortality of 36% compared with 15% for patients without an MR murmur. 56SAVE study investigators also showed that mild MR was associated with high mortality. Greater the severity of mitral regurgitation, worser was the prognosis.⁴⁸ Even uncorrected mild MR, as well as moderate to severe ischaemic MR is found to be associated with higher mortality on long term.

1However, the severity of ischaemic MR tends to follow the severity of the LV dysfunction causing the MR; the worse the MR, the worse the LV functions. Till date, **there are no studies**

to prove that ischaemic MR is a predictor of long term prognosis irrespective of severity of LV dysfunction present.⁸ No studies have proved that correction of mitral regurgitation will mitigates the long term mortality independent of left ventricular systolic dysfunction⁸ Though small case series has shown that coronary revascularisation in acute MI has reduced the severity of MR in selected patients, it tend to persist in > 50% of patients of MI on follow up.¹² Therefore, even coronary revascularisation may not halt its progression during long term. Prognosis is also found to be related to the dynamicity of component of ischaemic mitral regurgitation. A 5

1fold increase in the relative risk of death with an exercise-induced increase of ≥ 13 mm² of the ERO area

was found.^{8, 57} It is the best predictor of hospitalisation and cardiovascular morbidity as compared to severity of mitral regurgitation at rest. Its deleterious effects are related to several factors like sudden increase in R Vol (regurgitant volume) and with rapid QRS widening due to increases in

1ventricular wall stress leading to worsening **LV**

dyssynchrony.⁵⁸ In

1patients with LV systolic dysfunction, acute pulmonary oedema

which may develop due to sudden worsening of dynamic MR leading to acute increase in left atrial pressures.^{8, 59} Greater exercise-induced regurgitant volume and systolic pulmonary arterial pressure may cause exertional dyspnea.⁶⁰ Around 20% may have

1an improvement in the severity of ischaemic mitral regurgitation **during exercise,** who **have a** favourable **long term prognosis.**

⁵⁷ This is thought to happen

1in patients with contractile reserve in **posterior**

segment.²⁹ MANAGEMENT Numerous treatment options have been proposed but the treatment of ischaemic mitral regurgitation still remains a complex issue and needs further research and trials to find an ideal treatment modality with long term benefits. In current clinical approach at most places ring annuloplasty is preferred treatment modality. However, long term benefits of this technique remains obscured as it has no role in correction of local alteration due to left ventricular remodelling. Medical Management Standard anti-failure medications such as angiotensin-converting enzyme (ACEI) inhibitor or ARBs if the ACEI is not well tolerated), aldosterone antagonist and beta-blockers .^{8, 61} It may help in alleviating the severity of mitral regurgitation by producing reverse left ventricular remodeling.^{8, 62} Cardiac resynchronization therapy Though biventricular pacing per se is not an treatment modality for ischaemic mitral regurgitation, it is an indication to consider CRT in patients of ischemic MR with reduced left ventricular ejection fraction ,functional class III or more even with medical treatment and ecg showing QRS of more than 120 ms.⁶³ Cardiac resynchronization therapy resynchronize the papillary muscles and increase the closing force which helps in immediate reduction in MR.^{8,64- 65} There is further more reduction in the severity of MR in long term that is after few weeks or months as it plays a role in LV reverse remodelling, through a reduction in tethering .It can also reduce dynamic MR.⁶⁶ Magnitude of MR induced by exercise attenuate significantly in parallel to reverse Left Ventricular remodelling over a period of three months and result in improved cardiopulmonary performance.^{8, 67} Despite a reduction in the severity of MR, residual MR frequently persists. Immediate recurrence of MR has been seen post withdrawal of CRT due to dyssynchronization of the papillary muscles leads to. ⁶⁸⁻⁷⁰ Percutaneous coronary revascularization It may help in reducing the severity of mitral regurgitation at rest as well as during exertion in the subset where it is directly induced by ischaemia. .⁸ Surgical Management Surgical approach for management of ischemic MR can only reduce its severity, not eliminate it completely.

Coronary artery bypass grafting by itself is regarded insufficient in correction of MR. 71 Persistence of even little residual mitral regurgitation postoperatively has been shown to be associated with higher mortality 72 With the use of an undersized prosthetic (preferably two- sizes) ring 73,

1reduction in LV volume has been seen **and even a small increase in LV ejection fraction**

has been documented , 74 but the long-term benefits could not be proved.75,76 Several studies have shown that long-term outcomes in terms of survival benefit or functional outcome is questionable by combined surgery. 75,77-78 In a recent randomized control trial it has been shown that mitral valve repair done along with CABG was associated with improvement of NYHA class, LVEF, and reduction in left ventricular diameter, left atrial size and PAP(pulmonary artery pressure) .79It

1was not powered enough **to analyse the effect on**

mortality . To conclude it may be said that fixing some valves may help, but it is difficult to identify which ones.80 Recently published

1meta-analysis showed **that mitral valve repair for ischaemic** mitral regurgitation **is associated with better**

survival compared with MVR (mitral valve replacement). 81 The European Society of Cardiology

1guidelines recommend that patients with severe ischemic mitral regurgitation **(ERO area ≥ 20**

mm²) undergoing

1CABG should be treated by combined surgery (class I, level of evidence

C). 8,82Mitral valve repair may be considered

1in symptomatic patients with severe mitral regurgitation **who cannot be revascularized** is **questionable**

(class IIb).82 Mild mitral regurgitation should be managed conservatively. Due to the lacuna of well defined guidelines and evidence, the management of ischemic MR should be individualized. Assessment of

myocardial viability, especially in region of posterior basal wall, inducible ischemia and the dynamic component of MR aids in decision making. Biphasic response or regional contractile reserve during stress testing usually have exercise-induced reduction in mitral regurgitation, can be help decision making. Patients with exercise-induced increase in effective orifice area $\geq 13 \text{ mm}^2$ could be taken up for combined surgery. Severity of MR is always underestimated even with intra-operative TEE. Pharmacological measures like phenylephrine or rapid fluid challenge may be used to assess the ischaemic MR.⁸³ Parameters like grossly dilated left ventricle, multiple regurgitant jets, systolic sphericity index, wall motion score index, ESV(end-systolic volume), severe MR, $>2.5 \text{ cm}^2$ systolic tenting area, , large angle ($\geq 45^\circ$) of the posterior leaflet, $>1 \text{ cm}$ distance between coaptation point and mitral annulus are recognized as predictors of bad outcome of procedures like mitral valve repair by annuloplasty.⁸⁴⁻⁸⁶ Several adjunctive techniques have been proposed like chordal cutting, internal direct repositioning or external repositioning of the displaced papillary muscle.^{8, 87} However, they are not yet clinically approved for routine management of ischaemic mitral regurgitation. PERCUTANEOUS REAPIR Percutaneous edge-to-edge Alfieri procedure has been used for the treatment of MR due to either ischaemic or organic cause. In it, the central parts of both mitral leaflets are apposed producing a double orifice.^{8, 88} Many researchers have developed the devices which can be delivered in coronary sinus and reduce the severity of mitral regurgitation by pushing the PML forward.⁸⁹⁻⁹⁰ Long term effects of these devices needs to be studied. FORTHCOMING THERAPIES Future targets like transplanting autologous myoblast has potential proven to cause localized LV reverse remodelling and appears to be a promising approach in decreasing ischaemic MR. More understanding of our concepts of leaflet adaptation in LV dysfunction can help us in developing potentially better therapies in future.⁹¹ 3D EF Measurement in Myocardial Infarction Echocardiography is the most common method to assess left ventricular systolic function. It can efficiently predict the outcome and help in determining the treatment modalities like CRT-D implantation etc. Routine 2D EF measurement has several limitations in AMI patients due to problems of foreshortening and geometric assumptions. 3D echo LVEF is much more accurate especially in presence of regional wall-motion abnormalities as it does not have geometric assumptions and is found to have comparable with present day "gold standard" cardiac MRI.⁽⁵⁾ It can automatically calculate ejection fraction and left ventricular mass using the automated softwares, and is therefore more reproducible. It is up to 3 times more accurate than 2DE LVEF.⁷ Figure 7. Measurement of 3D EF in echocardiography through Q lab analysis DESIGN AND METHODOLOGY Study Design:

11 **This is a single centre** prospective observational **study**

done in the department of Cardiology, Christian Medical College (CMCH), Vellore. SETTING

11 **Study was** done **in the** Cardiology **department of Christian Medical College** and

Hospital, a tertiary care hospital in South India. 119 consecutive patients with STEMI were assessed for mitral regurgitation using various echocardiographic parameters. STUDY PARTICIPANTS Inclusion criteria: All patients older than 18 yrs old, who sustained STEMI between September 2011 to August 2012. Exclusion criteria: Patients were excluded from the study if they had 1. Rheumatic heart disease 2.

Persistent arrhythmias 3. Known coronary artery disease(CAD) 4. Cardiogenic shock 5. Trivial MR Methods:
 1. Demographic and clinical profile of patients were collected once they consented 2. All patients underwent echo once they were stable during index admission. Mitral regurgitation quantified using jet width[vena contracta], jet area

7and proximal isovelocity surface area(PISA)

,if central jet is present 3. Left ventricular

7ejection fraction were assessed by both Simpson's method

as well as 3D echocardiography STATISTICAL ANALYSIS Statistical analysis was done using commercially available statistical software ('IBM SPSS software version 15', Illinois, Chicago). All

10continuous variables are expressed as mean \pm SD and categorical variables are expressed as number (percentages). Independent samples T test was used for comparative analysis of

two groups with a normally distributed continuous variables. Comparison of categorical variables was done using Chi square test. Pearson correlation coefficient was used to analyze the correlation between two continuous variables with a normal distribution. Comparative

8analysis was performed by one way analysis of variance (ANOVA). A p value less than 0.05 was considered statistically significant for all test results.
 RESULTS 1. STUDY PROFILE AND

BASELINE CHARACTERISTICS

4A total of 119 patients of STEMI were evaluated

for ischaemic MR during the study period.

4Mean age of patients in the whole study population was

53.42 \pm 11.47 years. Majority patients (57.1%) were found to be in the age group of 40-59 years, with least number was in >80 yrs (2.5%).10.9% patients were young, <40 years having MI. Number of males outnumber within each group.Ratio of Males: Female is 96:23. 31.9%(38) of the patients has type 2

diabetes mellitus,98.3%(117) of patients had dyslipidemia, 37.8(45) % of the patients had hypertension ,54.6% (65) were obese , 47.1%(56) were current smoker and 13.4%(16) had positive family history of coronary artery disease(CAD). On an average all patients were had at least 2 risk factors. Baseline characteristics of both groups with Anterior wall MI (AWMI) and Inferior wall MI (IWMI) were comparable without any significant statistical difference except baseline 2D EF, which was higher in IWMI group (table 1). Patients presenting with AWMI were having slightly higher proportion of hypertension, though it was statistically insignificant. Characteristic AWMI IWMI p value Age 52.30±11.11 54.58±11.81 .281 Male 47(78.3%) 49(83.1%) .515 Female 13(21.7%) 10(16.9%) .515 Diabetes 19(31.7%) 19(32.2%) .85 Hypertension 27(45%) 18(30.5%) .103 Smoking 29(48.3%) 27(45.8%) .779 Dyslipidemia 59(98.3%) 58(98.3%) .990 Obesity 35(58.3%) 30(53.1%) .293 Positive Family History 10(16.7%) 6(10.2%) .299 Baseline 2D EF 44.94±7.5 50.7±7.4 .000(significant) Baseline 3D EF 45.42±8.5 46.96±8.6 .33 Table 1.Baseline characteristics of patients according to the type of STEMI 2. Prevalence of MR 27(22.7%) patients were found to have MR during index admission for MI (Figure 8), out of which 24(88.9%) had mild MR and 3(11.1%) had moderate MR based on quantitative measurements. None of the patients were found to have severe MR. Mean Vena contracta (VC) in mild and moderate MR patients at presentation were 2.91±0.94 and 3.54±0.25 respectively. Mean PISA radius, effective regurgitant orifice area (ERO) and regurgitation volume in mild MR patients at presentation were 0.315±0.08, 0.05±0.03 and 7.07±3.65 respectively (table 2). MR VC(mm) PISA ERO(cm²) MR radius(cm) Volume(ml) MILD(n=24) 2.91±0.94 0.315±0.08 0.05±0.03 7.07±3.65 MODERATE(n=3) 3.54±0.25 0.44±0.06 0.10±0.06 30.33±7.77 AFTER 1 MONTH MILD(n=12) 2.97±0.55 0.38±0.81 0.05±0.03 6.9±3.17 MODERATE(n=2) 4.8±8.5 0.55±0.07 0.125±0.02 27±1.41 Table 2.Quantitative echocardiographic parameters of MR at presentation and after 1 month Mean age of patients presenting with ischaemic MR at onset was 59.2 ± 10.82(figure 9), population includes 74.1% males (20) and 25.9 % (7) females. Majority of patients presenting with MR at admission were in the age group of 60-79 years (figure 9).Among the patients having MR at admission included 48.1%(13) type 2 diabetics, 100%(27) dyslipidemics, 33.3 % (9) hypertensive's, 44.4% (12) obese, 44.4 % (12) current smoker, 22.2%(6) had AWMI, 77.8%(21) had IWMI and none had a positive family

4history of coronary artery disease (CAD).

Though there was trend towards females being more commonly associated with MR, it was found to be statistically insignificant (figure 10). None of the risk factors predicted of onset of MR in STEMI patients at admission and during follow up on statistical analysis except age, diabetes and site of STEMI(Table 3).IWMI patients were more likely to develop MR at presentation (table 3).Male gender was found to have higher prevalence of absence of MR during follow up. MR 22.7 PRESENT 77.3 ABSENT Figure 8.Prevalence of MR at admission 60 Number of STEMI patients 50 40 30 MR ABSENT 20 MR PRESENT 10 0 20-39 40-59 60-79 >80 Age groups Figure 9.Prevalence of MR according to the age groups 100 100 90 80 79.2 77.8 78.6 76.1 80 70 65.2 65.8 60 50 40 30 20 20.8 34.8 34.2 22.2 21.4 23.9 20 PRESENT ABSENT 10 0 0 Figure 10.Proportion of baseline risk factors of patients with and without MR During follow up after 1 month, MR was found to be present in 15.7% of patients despite a dropout of 30(25.2%) patients (figure 11).MR continue to persist in 10.1 % (9) patients with improvement in severity in 13.5 % (12) and new onset in 5.6 % (5) patients (figure 12).Among 14 patients who were having MR at follow up, 3(21.4%) had AWMI and 11(78.6%) had IWMI at baseline. None of the baseline characteristic was found to be predictor of mitral regurgitation during follow up (table 3) excluding sex and site of STEMI.AWMI and

males tend to have lesser prevalence of mitral regurgitation at follow up (table 4). IWMI patients continue to have higher incidence of Mitral Regurgitation which was persistent and also developed new onset MR, but it was not statistically significant ($p=0.647$). Females have higher prevalence of Mitral Regurgitation during follow up visit (table 3). None of the patients with positive family history had Mitral Regurgitation at onset. Development of MR in STEMI patients was not influenced by the treatment modality used (figure 13). MR

15.7 84.3 ABSENT PRESENT Figure 11. Prevalence of MR after 1 month 14 13.5 12 10.1 Number of patients 10 8 6 4 5.6 2 0 MR Figure 12. Profile of MR after 1 month Proportion of patients having greater severity of MR after 1 month was found to be slightly higher, despite being reduction in absolute number of patients having moderate MR (figure 14). Initially, 3 (11.1%) patients had moderate MR, but at 1 month, only 2 patients had moderate MR. IWMI patients were found to have higher prevalence of MR (figure 15). 60 51 50 Number of patients 40 31 MR PRESENT 30 MR ABSENT 20 11 14 10 10 2 0 THROMBOLYSIS PRIMARY PTCA OTHERS Figure 13. Prevalence of MR in relation to treatment strategies Characteristic Baseline MR p value MR at 1 p value month Age (yrs) Group 1 (20-39) 1(3.7) 2(14.3) Group 2 (40-59) 11(40.7) 0.024* 5(35.7) 0.132 Group 3 (60-79) 14(51.9) 7(50) Group 4 (≥ 80) 1(3.7) 0(0) Sex Male(96) Female(23) 20(74.1) 7(25.9) 0.323 9(64.3) 5(35.7) 0.007* Site of STEMI AWM(60) IWMI(59) 6(22.2) 21(77.8) 0.001* 3(21.4) 11(78.6) 0.03* Diabetes($n=38$) 13(48.1) 0.04* 5(35.7) 0.612 Hypertension($n=45$) 9(33.3) 0.585 4(28.6) 0.615 Smoking($n=56$) 12(44.4) 0.757 5(35.7) 0.210 Dyslipidemia($n=117$) 27(100) 0.44 14(100) 0.539 Obesity($n=65$) 12(44.4) 0.36 5(35.7) 0.257 Positive Family History($n=16$) 0(0) 0.02* 1(7.1) 0.458 Baseline 2D EF 48.17 ± 8.93 50.2 ± 8.6 Baseline 3D EF 46.24 ± 7.68 50.37 ± 8.61 0.216 Reperfusion modality Thrombolysis ($n=45$) Primary PCI ($n=62$) Misc($n=12$) 11(40.7) 14(51.9) 2(7.4) 0.744 8(57.1) 6(42.9) 0(0) 0.134 Table 3. Comparison of various variables with MR at onset and during follow up

statistically significant MR Sex Total p value 1(Male) 2(Female) Absent Number of patients 69 7 76 (88.5%) (58.3%) 84.4% 0.019 Present Number of patients 9 5 14 (11.5%) (41.7%) 15.6% Total Total no. 78 12 90 % within sex 100.0% 100.0% 100.0% Table 4. Association of sex with the prevalence of Mitral Regurgitation at 1 month Severity Baseline MR MR at FU Mild 24(88.9) 12(85.7) Moderate 3(11.1) 2(14.2) Severe - - Figure 14. Prevalence of severity of MR at onset and after 1 month 100% 90% 80% 70% 38 60% 53 MR ABSENT 50% MR PRESENT 40% 30% 20% 21 10% 7 0% AWM IWMI Figure 15. Prevalence of MR in relation to type of MI 3. Correlation among 2D and 3D EF in STEMI Mean 2D EF in AWM group at presentation was $44.94\% \pm 7.5\%$ and in IWMI group was slightly higher $50.7\% \pm 7.4\%$. LV systolic functions was found to be better in IWMI patients on presentation. After 1 month, there was an increase in 2D EF by 6.9% (51.83 ± 8.06) in AWM group and negligible change in IWMI group (51.05 ± 8.4). 2D EF was found to strongly correlate with 3D EF at admission and during follow up irrespective of type of STEMI (table 5), though poor image quality was one of the hindering factor in some of the patients. Figure 14 shows the scatter plot displaying the distribution of 2D and 3D EF at baseline. 2D and 3D EF Correlation p (2 tailed) coefficient, r At admission 0.525 0.01 At 1 month 0.609

40.01 Table 5. Correlations of 2D and 3D

EF at admission and after 1 month $70 y = 0.5651x + 19.17960$ $R^2 = 0.2756$ 50 Baseline 3D

14 EF 40 30 20 10 0 0 10 20 30 40 50 60

70 80 Baseline 2D EF Figure 16. Scatter plot showing relationship of 2D-EF and 3D-EF at admission (Pearson correlation coefficient r , 0.525) *

12 Correlation is significant at the 0.01 level (2-tailed). DISCUSSION The present study

is a prospective observational study which evaluated the prevalence of functional (ischaemic) mitral regurgitation in patients with STEMI at presentation and during 1 month of follow up.

4 Mean age of patients was 53.42 ± 11.47 years

with majority of them being males (83%). Most common age group was found to be 40-59 years (57.1%), with least patients in >80 yrs (2.5%). 10.9% patients had MI in young (<40 years). Our results are found to be similar as observed in a large retrospective analysis by Brijesh et al⁹² done in our institute. Most common risk factor was found to be dyslipidemia (98.3%), followed by obesity (54.6%), smoking (47.1%), diabetes (31.9%), hypertension (37.8%) and positive family history of coronary artery disease (13.4%). This data is different from create study where diabetics 34% had diabetes; 37.7% had hypertension; and 40.2% were smoker.⁹³ All patients were having multiple coronary risk factors. LV systolic function was found to be better in IWMI patients at presentation as lesser territory was compromised by ischaemia. Similar results were observed by Darbar et al and our findings are consistent with it.⁹⁴ With anterior infarction, the injury is exclusively in the left ventricle, whereas inferior infarction is associated with injury to both ventricles, causing less impairment of left ventricular function despite an equivalent overall myocardial insult. After 1 month, there was a trend in the increase in 2D EF by 6.9% in AWMI group due to revascularisation therapy.⁹⁵ MR was found in 22.7% at presentation of STEMI in our study population, which is in accordance with older studies including angiographic ones (table 5). It was found to be higher in older age group, diabetics and IWMI patients at the onset, which was consistent with previous studies.^{2, 12, 15} There was a trend toward an excess of women in the MR group, though it was not statistically significant due to under representation of females in present study. Similar data on the demographics of MR after AMI have been presented earlier by Lehmann, Tchong, and Barzilai.^{2, 12, 15} IWMI involves the posterior wall of LV leading to left ventricle remodelling and distortion. This result in apico-lateral as well as posterior displacement of papillary muscle, which in turn leads to apical displacement of mitral leaflet coaptation point, producing the ischaemic mitral regurgitation.⁸ Mostly patients had mild MR and none had severe MR which is possibly due to exclusion of very sick patients like cardiogenic shock. During follow up after 1 month, MR was found to be in 15.7% of patients despite a dropout of 30 (25.2%) patients. Some patients with MR tend to improve with the resolution of acute ischaemia due to revascularisation as well as favourable LV remodelling and improvement in LV systolic function, while some had deteriorated with worsening mitral regurgitation due to adverse left ventricular remodelling with passage of time after STEMI. Treatment modality used didn't influence the development of MR as shown earlier by Tchong JE et al. Acute reperfusion with thrombolysis or angioplasty did not reliably reverse valvular incompetence as seen in earlier observational studies. None of the baseline risk factor except diabetes, territory involved and older age group, can reliably predict the development of MR at onset and left ventricle systolic function, though this study was not powered enough to study those predictors. IWMI patients tend to have higher



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INTRODUCTION MR is a frequent complication of coronary artery disease. It was often under-rated because it is clinically silent, but with the use of echocardiography this complication is observed between 15%-20% after a myocardial infarction.¹ When compared with patients without MR, the patients with acute myocardial infarction and mitral regurgitation are older, more often in female and frequently have a history of previous ischaemic heart disease.² Its presence and degree have major prognostic implications and underscore the importance of its detection and quantification. Ischaemic mitral regurgitation can independently predict cardiovascular death with a relative risk of 2.3 In a...

prevalence of MR during the STEMI as well as during follow up secondary to tethering effect on the mitral valve leaflet which may be a harbinger of chronic MR later on. Treatment modality didn't influence the severity of MR in STEMI patients. Author Year No of Modality used Prevalence patients of MR (%) Barzilai B et al¹⁵ Tchong JE et al¹² 1988 1480 Echo 39 Lehmann KG et al² 1992 1480 Echo 17.9 1992 206 LV ventriculography 13 Gervasio A. Lamas et al³ 1997 727 LV ventriculography 19.4 Present study 2013 119 Echo 22.7 2D EF was found to correlate linearly with 3D EF irrespective of type of STEMI. In a recently published metanalysis, Jennifer L. Dorosz et al⁹⁶ showed similar results. With EF, there is no difference in the bias between 3DE and 2DE, and the difference in the variance is modest ($\pm 4.7\%$). Despite being subjected to errors due to foreshortening, poor endocardial definition, narrow echocardiographic windows, and assumptions about LV shape, it remains a ubiquitous tool for assessing LV size and systolic function in day to day clinical practise. Although 3DE shows promise in providing the accessibility of echocardiography and the multi- planar imaging of CMR, this nascent technology still has limited spatial and temporal resolution compared with gold standard modality of CMR. Despite its limitations, 3DE may be superior to 2D techniques. Our single centre study shades light on the prevalence of Mitral Regurgitation in acute STEMI in Indian population. MI is most prevalent in middle age persons with ischaemic Mitral Regurgitation in slightly older age group. Modalities of revascularisation (mechanical vs thrombolytic or medical treatment doesn't influence the course of Mitral Regurgitation in short term follow up. However, these findings need to be confirmed in larger study population MI. STUDY LIMITATIONS The main limitation of this study was smaller sample size, which was not powered enough to study the predictors of development of MR in STEMI patients. Moreover, females were underrepresented in the study group, which may have negated the gender effect on ischemic MR. Critically ill patients , cardiogenic shock etc were not included in whom ,there is a probability of having severe mechanical complications of STEMI like severe MR. There was a significant drop out during follow up, which may have influenced the statistical analysis. Only echocardiographic LV systolic function measurements were used and were not compared with other imaging modalities like MRI, which is gold standard for determining EF in patients with regional wall motion abnormalities. Longer follow up periods are required for assessing the development of chronic ischaemic MR. CONCLUSION STEMI patients have 22.7% prevalence of MR at onset, which is more likely in older age group, diabetics and IVMI patients and independent of gender and left ventricle systolic dysfunction. Despite under-representation of females in our study population, they seem to carry higher risk of development of ischaemic Mitral Regurgitation in India. Most commonly, ischaemic MR is of milder severity at onset which tends to persist in 15.7% of patients during follow up. There is a significant association of development of ischaemic MR during acute STEMI with IVMI. Some patients may develop new MR later on depending on the LV remodelling. 2D EF estimation with a properly acquired good image is comparable to more precise 3D EF in STEMI patients. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50 51 52 53